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BULLETIN

OF THE NEW YORK ACADEMY OF MEDICINE

Original Articles by

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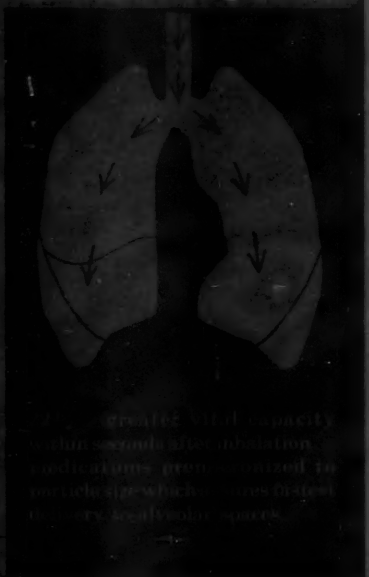

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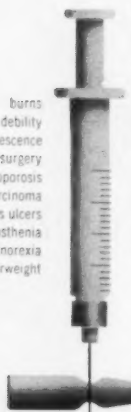

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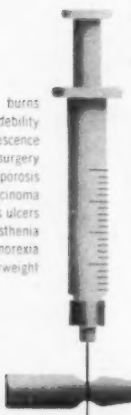

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*Miller, J.B., et al.: Ann. Allergy, 12:611, Sept.-Oct., 1954.

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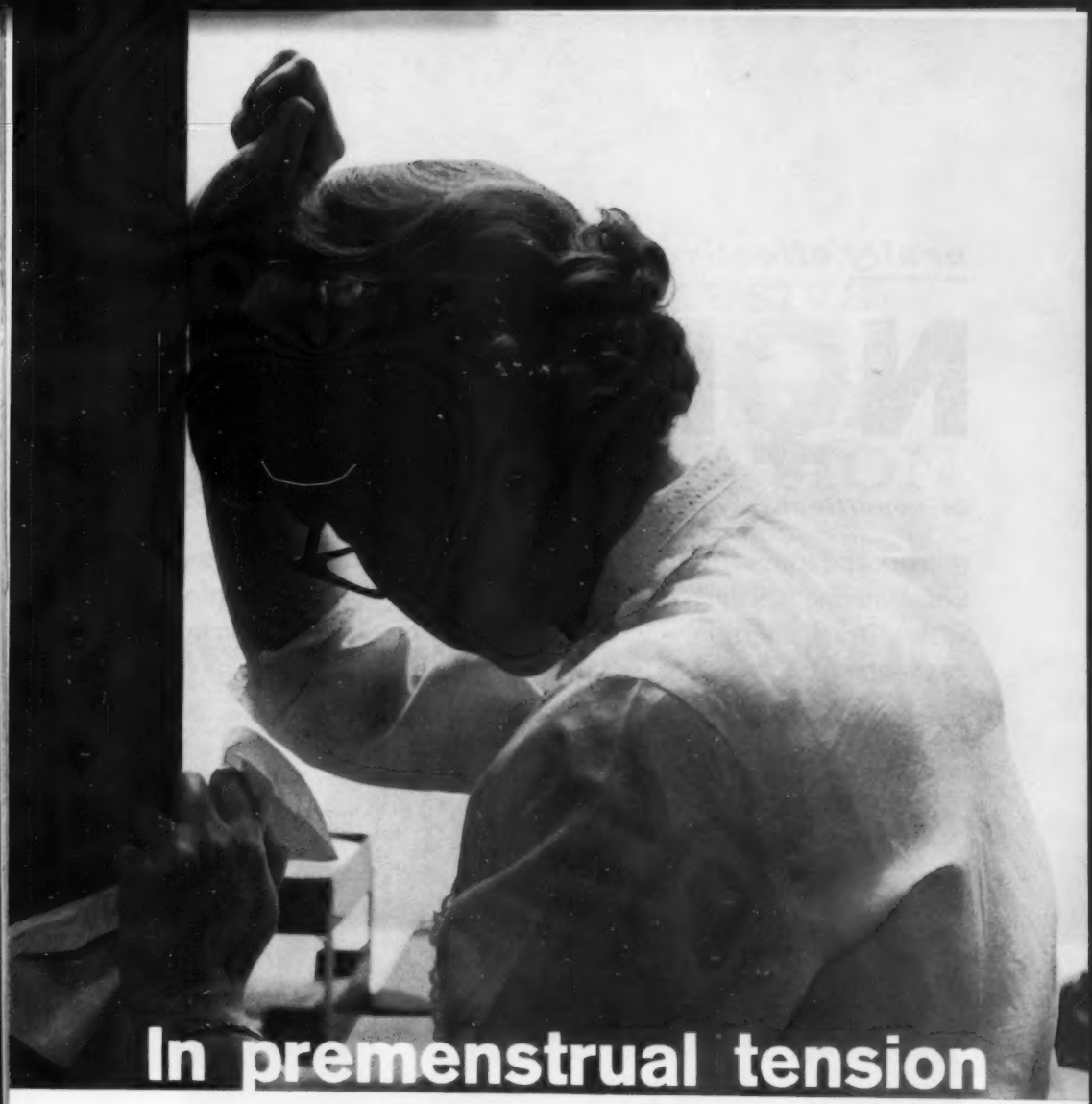

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**Podolsky, E.: Journal-Lancet 79:318 (July) 1959.*

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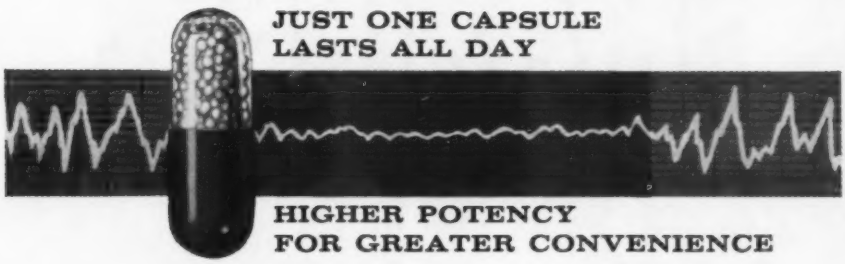

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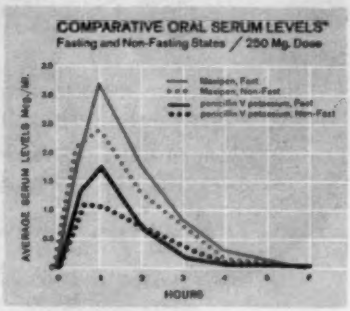

*Based on 3294 individual serum antibiotic determinations. Complete details available on request.

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1. A. M. A. Arch. Int. Med., *99*:346, 1957.
2. Am. J. Obst. & Gynec., *70*:1309, 1955.
3. Lancet, *1*:448, 1957.

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AUTHORS ALONE ARE RESPONSIBLE FOR OPINIONS EXPRESSED IN THEIR CONTRIBUTIONS

ROBERT L. CRAIG, *Editor*

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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

VOL. 36, NO. 4 APRIL 1960

THE ACADEMY'S PROGRESS

*Report of the President**

ROBERT L. LEVY

THIS has been a year of interesting developments at the Academy. A full account of them will appear in the next Annual Report. For the present review, various members of the full-time executive staff submitted summaries of the happenings in their respective domains, and I have abstracted this material. In presenting it, I shall be guided by the thought expressed in a couplet taken from Pope's *Essay on Criticism:*

"Launch not beyond your depth, but be discreet,
And mark that point where sense and dulness meet."

ABSTRACTS

The Director—Dr. Howard Reid Craig. The budget for 1959 was set up anticipating an income of $683,000.00, which is an increase of 3.3 per cent over 1958. This percentage is well below that published as indicative of the year's increase in national gross product and income. Greater costs of materials and labor are unavoidable. The high calibre of the staff must be maintained, and this can be accomplished only by paying adequate salaries. To meet such added expenses in the future, the Academy will be obliged to raise more money through contributions

* Presented at the Annual Meeting of The New York Academy of Medicine, January 7, 1960.

for general purposes. Any deficit for 1959 will be amply covered by reserves.

A lay advisory Finance Committee will be secured shortly to advise the Finance Committee of the Trustees regarding investment policies. The growth of the Academy's financial picture demands that such expert counsel be available on a continuing basis.

Following the retirement of Mr. Arthur A. Eberle as Comptroller, Mr. Robert M. Brereton was installed as Business Manager. He has already put into effect many of the recommendations concerning internal business management, as suggested in the recent Survey Report. There is great need for replacement of outworn furnishings and mechanical equipment. Changes have been made in personnel and responsibilities to secure greater efficiency in operation.

Major changes in organization also have been effected. Dr. Robert L. Craig, formerly Executive Secretary of the Committee on Medical Education and Editor of the *Bulletin*, has been placed in charge, as Executive Secretary, of all Academy publications and medical information services. Dr. Aims C. McGuinness has come to the Academy as Executive Secretary of the Committee on Medical Education and, in addition to the traditional functions of this office, will be responsible for coordinating the activities of the Committee with those of the Sections, and also with the work of the medical schools and hospitals throughout the city. There should result better development of New York's resources for graduate medical education.

A new division of press and public relations, and of fund-raising, has been established with Mr. Thomas L. Stapleton in charge. Such an arrangement centralizes all of these activities in one office.

These reallocations of responsibilities make the present Committee on Medical Information free to assume the title of Committee on Special Studies, with Dr. Iago Galdston continuing as its Executive Secretary. This group will pursue the projects which have become its major interest, such as the "Whither Medicine" study, and will continue to sponsor various symposia and conferences.

Mr. Keyes Metcalf, Librarian and Professor Emeritus of the Harvard University Library System, was engaged to undertake a detailed survey of the Library, to insure maximal efficiency and economy. His report will soon be forthcoming.

Through anonymous contributions and with the help of two of the

wives of Fellows, the east room off the main foyer has been redecorated and refurnished, and now serves as a lounge and reception room. This has added to the convenience of the Fellows and the appearance of the building.

Last year, plans for a "Deposit Library" were discussed. The proposed name has been discarded in favor of "The Medical Library Center of New York", a designation which connotes an actively functioning unit rather than merely a place for the storage of less used library material. Considerable progress has been made in arrangements for its establishment. A charter for a new corporation has been granted by the New York State Board of Regents under the Education Law of the State, the incorporators being the Deans of the five medical schools in Manhattan and the Bronx, the Clinical Director of Memorial Center, the Vice-President of the Rockefeller Institute and the Director of the Academy. Interest in the project has been shown by several foundations and certain grants already have been made. Negotiations for the purchase of a building to house this library are under way. It is reasonable to believe that, within a year's time, the Medical Library Center will be a reality.

The Academy occupies an important position in the community. It must maintain its services to the medical profession and the public. Upon the Fellowship itself rests the responsibility for assuring its financial security.

The Library—Miss Gertrude L. Annan, Librarian. The most important question in 1959 was concerned with the establishment of a cooperative Library Center. Reference to this matter has been made in the report of the Director.

The staff bent its efforts especially toward clearing away the various backlogs which have loomed large for some years. The frightening backlog in the Periodicals Department has dwindled to a few shelves of Russian periodicals. The catalogue backlog, however, remains, although progress in this area, too, has been made.

During the past year, work was begun on a staff manual which long has been badly needed. When completed, this will clarify work and, in many instances, simplify and modify library routines.

An essential step in making the contents of a library available is to make sure that its holdings are included in any relevant checklist

or bibliography. To that end, considerable time has been spent on three projects: a checklist of medical Americana through 1820; a bibliography of American imprints, 1801-1820; and a proposed guide to the location of American trade catalogues through the nineteenth century.

Nine exhibits have been prepared by the staff, ranging, in subject matter, from Herbals of five centuries to recent textbooks on pediatrics.

In 1955, the Library Publication Committee issued the first volume of a translation, into English, of the surgery of Theodoric. This important medieval text was translated from the Latin by the late Dr. Eldridge Campbell and Mr. James Colton, of Albany. The second volume has now been successfully completed and has been published by Appleton-Century-Crofts.

As usual, the Friends of the Rare Book Room were responsible for other long-sought texts. Undoubtedly, the most exciting acquisition in 1959 is a fine copy of the first edition of Peter Lowe's *The Whole Course of Chirurgerie*, published in London in 1597. This is an unusually scarce and valuable work and contains what is probably the first reference, in English, to ligation of arteries in amputation.

The Committee on Public Health—Dr. Harry D. Kruse, Executive Secretary. Pharmaceutical advertising was a major topic of concern to the Committee in 1959, as in 1958. The New York City Department of Health, pursuing a study made in the previous year, requested advice on possible measures to correct and prevent the faults found in the promotional material disseminated by pharmaceutical firms by direct mail to physicians.

The 1958 study confirmed statements made by the Committee in its report on tranquilizers several years ago, namely, that some of this kind of literature recommended products in such a way as to lead to, if not to encourage, indiscriminate use, and also that the material contained little or no mention of contraindications or side effects.

As a first step toward corrective measures, it was proposed this year that physicians be asked to report to the Department untoward reactions from drugs. With such a system of reporting, it may alert physicians to possible dangers in new drugs and prevent harmful effects. The Committee approved this plan and urged that the data gained from it be disseminated promptly.

The Department requested advice also on the more general problem

of misleading advertising. At the base of the question lies an inadequacy in procedures for the collection of data on new drugs, as evidenced by the usual history of such preparations. The full effects are seldom revealed until the drugs have been used extensively over a period of time. After some months, or even longer, reports of therapeutic limitations and adverse reactions appear. In some instances drugs have been withdrawn from the market. This sequence of events can be attributed to inadequate controls and to the use of investigators not trained in experimental therapeutics and evaluative technics.

Another aspect pertains to the literature distributed to physicians. It may be misleading in two ways: first, by overenthusiastic statements as to efficacy; and second, by omitting or giving insufficient space to side effects. Such effects constitute a hazard to the health of the public.

The remedy lies in a combination of education and legislation. The proposed system of reporting reactions would form a part of an educational approach. Another method would be to urge physicians to obtain detailed information about new drugs. A circular containing the information required by the Food and Drug Administration prior to its approval of new products is enclosed with the original packages delivered to pharmacists, but these the physician seldom sees.

The Committee urged that pharmaceutical firms set up an advertising code to discourage misrepresentations and to insist on proper accounts of both efficacy and undesirable effects. Individually and informally, some manufacturers appear to have adopted criteria for their advertising. Besides voluntary action, official regulations specifying minimum standards for the entire industry would be helpful.

The Committee's report on air pollution, published in the August issue of the Academy *Bulletin*, called for studies to throw more light on the effects, on health, of dust and fume-laden air. Although knowledge of these effects is still imperfect, enough is already known to warrant corrective measures.

Strong support for these views came from the New York State Commissioner of Health, Dr. Herman E. Hilleboe, who declared, in a recent statement, that air pollution is a health problem of serious proportions in New York State. He emphasized that, in the past ten years, the nature and extent of air pollution have changed radically, mainly because of the increased use of toxic substances in industry. Thus, it has become a complex problem requiring concerted effort by highly

trained professionals, buttressed by vigorous, sound legislation and adequate funds. In his opinion, it would be "dangerous folly" to await the establishment of cause-effect relationships.

For several years the Committee has been participating in a study of convalescent care in New York City, jointly with the Hospital Council of Greater New York and the United Hospital Fund. It was deemed desirable to survey all the convalescent facilities serving New York in order to establish present needs. In 1955 a grant was made to the School of Public Health and Administrative Medicine of Columbia University to carry out the project, under the supervision of a joint committee representing the three sponsoring organizations. The study is now virtually complete and it is anticipated that the final report will soon be ready.

Last month, the Committee reaffirmed its position approving fluoridation of the City's water supply. Letters to this effect were sent to the newspapers, to the Mayor and to the members of the Board of Estimate. Up to the present, a vote on the matter has not been taken by the Board.

The Committee on Medical Education—Dr. Robert L. Craig, Executive Secretary. The Committee arranged the programs of the Stated Meetings and of the Monthly Panel Meetings for the general physician. The Third Annual Postgraduate Week was entitled "Research Contributions to Clinical Practice"; the general format was similar to that of the past two years.

The third B. S. Oppenheimer Lecture was given by Dr. C. Walton Lillehei, Professor of Surgery at the University of Minnesota, who spoke on "Open Heart Surgery for Acquired and Congenital Heart Disease".

In October of last year, the National Institute of Mental Health gave a grant in support of a course in elementary psychiatry for general practitioners. This will begin early in 1960.

The Academy is acting as co-sponsor of a course on "Cardiovascular Diseases in Industry", arranged by the New York Heart Association. These exercises were started in November, 1959, and will continue through mid-February of 1960.

An *ad hoc* Subcommittee has been studying the effects of professional fee insurance on resident teaching programs in voluntary hospitals. The report of the Subcommittee has been completed and approved by the Council.

The Committee on Medical Information—Dr. Iago Galdston, Executive Secretary. Many inquiries were received and dealt with and a number of releases were issued. Numerous manuscripts were submitted for review by the major national weekly publications.

The "Whither Medicine Pilot Study" held eighteen meetings, at which addresses were given by guest speakers, on a variety of topics. Additional funds for continuing the study were granted by the Commonwealth Fund. Four monographs dealing with various aspects of this project will be issued.

The Committee published two books—*Medicine and Anthropology* and *Lectures to the Laity, Series 19 and 20.*

The weekly Postgraduate FM Radio Program, "For Doctors Only", has been presented throughout the year over Station WNYC.

"On Medical Frontiers" is the title of the twenty-fifth series of "Lectures to the Laity", organized for the 1959-1960 season.

The Eastern States Health Education Conference was again held at the Academy. The theme was, "A Critique of the Psychology of Health Education".

The Bulletin—Dr. Robert L. Craig, Editor. The *Bulletin*, issued monthly, has continued to publish original articles which have been presented at Academy meetings, as well as transcripts of panel meetings and abstracts of papers given at scientific sessions of the New York Pathological Society and of the New York Heart Association. In addition to being distributed to all Fellows, the *Bulletin* goes to 1325 paid subscribers, of whom 371 are in foreign countries.

Epilogue

Yesterday was the one hundred and thirteenth birthday of the Academy and, during its lifetime, many traditions have been established. It is these which give flavor and character to an organization; their perpetuation reflects pride in its past. But traditions, by themselves, do not suffice to further thought and knowledge. From time to time, new stimuli are required to initiate advance; to stand still is to regress.

In a rapidly changing environment, the goals toward which we strive undergo frequent modification. Ideals are rarely, if ever, fully realized; yet their existence should never be forgotten. To approximate them as closely as possible will be the Academy's continuing aim.

SCIENCE, POLITICS AND PEACE*

The Anniversary Discourse

DR. GRAYSON KIRK

President of Columbia University

A WEEK AGO the decade of the Fifties came to a close. In time, historians may be tempted to attach some distinctive label to these past ten years, and it is amusing to speculate how our grandchildren may be prompted to characterize the years through which we have just passed. Such glib generalizations frequently involve alliteration, so it is quite possible that future generations may talk knowingly about life in the United States during the "Fumbling Fifties".

At the moment, and without the perspective of hindsight, this adjective seems uncomfortably accurate. We have passed through a decade of marked confusion, a decade when old and familiar foundations of both our national society and our international life seemed to be under attack by forces that were ominous, forces that were so new and unprecedented in our experience that they could not be estimated by any traditional rules or principles. Both in domestic and foreign affairs we have had the conviction that we have been thrust unaccountably into a new world, not at all a brave new world, but one that engenders fear because its prospect has left us with a feeling that we may not be able to understand and master it in the time that may be allotted to us. For the first time in our history, we do not feel that we can take the future for granted.

Our sense of disquiet has been reenforced, not merely by the emergence of the great new revolutionary forces that have suddenly swept across the world, but also by what appears to be an ever-accelerating rate of change. Before we have had time to grapple with a new situation, to understand it and to chart an appropriate course of

* Given at the Annual Meeting of The New York Academy of Medicine, January 7, 1960.

action, we have had to cope with a dozen other new situations whose complexities and interrelations have baffled even our best minds. We have wondered whether, in Toynbee's phrase, we were entering into a "time of troubles".

Our concerns have not been exclusively with the outside world and its menace. We have had the feeling that, at home, unhealthy influences have been growing in an alarming fashion. Old institutions and attitudes traditionally fostering a sense of unity and stability today seem less vigorous and effective. We observe with alarm the rising incidence of crime, the shocking growth of juvenile delinquency, the declining level of individual and group morality—all these trends that are dismally familiar to any citizen whose morning newspaper brings him the record of the day before. There are times when it seems that the concern of the American public for the general welfare has been replaced by a new criterion of conduct aptly if inelegantly expressed by the question, "What's in it for me?". In a society where "getting by" is an accepted canon of conduct, where the cardinal sin is in being found out, notions of morality become vague; "good" and "evil" become only relative terms. Sometimes it appears as if we are more concerned with understanding the motivation of the criminal or the delinquent than in condemning his acts or in protecting ourselves from them.

Admittedly, this is but one facet of contemporary society. It must be balanced off by all the great forward strides that we are taking, none of which is more striking than in the field which this Academy so well serves. Nevertheless, we do ourselves a great harm if we become so obsessed with our progress that we overlook or underestimate the menace of these influences and trends that eat like termites into the solid foundations of a healthy society. It is but the judgment of a single individual, but for what it is worth, it is my belief that these corrupting forces are allowed to operate under conditions of greater public apathy than ever before. It is also my belief that we now face a time when we shall have need of all our spiritual vigor to meet successfully the fearsome problems of the world of the nineteen sixties.

As we look at the outside world our perplexity is even greater. Within the span of a few years our nation's vast geographic barriers, behind which we grew safely to great-power status, have vanished. We now stand naked and exposed to foreign military dangers that we thought we would never be obliged to face. Yesterday we thought that

non-involvement in world affairs would protect us. Today we have military installations scattered more or less permanently over a large part of the earth's surface. Yet despite this vast and expensive effort to protect ourselves, we live under the menace of unimaginable destruction that could be brought to us by impersonal instruments dispatched from launching pads half way round the world. Yesterday we were unconcerned, in the matter of security, with political or economic events in most of the world. Today, in remote Tibet, the jungles of Africa, or the tiniest islands off the Asian mainland, every economic or political crisis is a matter of security concern to us.

It is not merely in the matter of military security that our world has so suddenly changed. Traditionally, we have not questioned the belief that, in due time, the remainder of the world gradually would embrace at least the essential features of our economic and political order. We were so confident of their inherent superiority that we did not see how the trend of history could be otherwise. We were convinced that, given freedom of choice, all peoples would prefer liberal democracy and economic free enterprise. We were sure that these twin principles were essential to political stability and sound economic progress manifested by an ever-rising standard of living. We thought that authoritarian systems of government were unstable because they tended to be unenlightened even when they were not corrupt. Only in the friendly climate of free enterprise could industrialization be rapid and efficient.

Today we have not weakened in our fidelity to these principles at home, but we are confused because they seem to be faring so badly elsewhere in the world. Postwar liberal democracies set up in many newly independent countries have given way, one by one, to more authoritarian regimes. Where liberal democracies still persist, they are characterized all-too-frequently by conditions of political and economic instability or even near-chaos. As we have observed the results when a stronger hand of authority has quelled disorder, improved public morality and fostered economic stability, we know that we would be doctrinaire indeed if we were to condemn what has happened.

Further, it is impossible to be oblivious to the fact that some of these authoritarian regimes have mobilized the human and other resources of their people to bring about a rapidity of industrialization that we had thought impossible. For a time we were unimpressed by these unfore-

seen developments. As we watched what was happening in Soviet Russia, and more recently in China, we reassured ourselves that bureaucracy, the lack of highly trained technologists, and the inertia of a partially or wholly coerced labor force would doom the enterprise to a lumbering inefficiency and to end-products of inferior quality. Today, as we watch the spectacular achievements of Soviet science and engineering orbit through the skies, we realize that this was at best a premise of short-run validity, much shorter than we had thought or hoped.

Until recently we clung to the belief that all non-democratic regimes would be transitory. We thought that they would be swept away by internal collapse and the revolt of peoples who refused to live under regimes that gave them so little freedom. Now we are being forced to admit that many of these governments appear to have grown in stability and in popularity. It seems increasingly clear that while severe limitations upon freedom continue to exist, at least some of the peoples who live under these authoritarian regimes seem to have accepted this absence of freedom as a bargain price to pay for a high rate of economic growth and progress. Perhaps we have overlooked the fact that all peoples, no matter what the nature of the political regime under which they live, develop a sense of pride in national achievement. Perhaps we forgot that peoples can be incited to great exertion—and to great sacrifice—for a cause that transcends considerations of unfamiliar personal liberty or individual profit. At any rate, whatever the sources of our error, we are now obliged to conclude that many of these authoritarian regimes seem likely to last into an unforseeable future, and that they will last because their use of both the carrot and the stick is accepted by the majority of the citizens. The notion that every authoritarian regime is inescapably a slave state in which the great majority of the people groan under a yoke they will throw off at the first opportunity—this is wishful thinking, and we are at last beginning to realize it. We now know that we are destined to live indefinitely in a world where significant numbers of states and peoples will not have economic or political institutions like our own; where, in fact, they will reject both free enterprise and the principle of legal and protected political opposition. This is to be part of our new world.

Our confusion over the revolutionary tumult of the last decade is heightened by our realization that the so-called "underdeveloped" peo-

ples of the world, now largely free from colonial rule, are so determined to achieve rapidly rising living standards that they will accept the economic and political system that seems most likely to give it to them in the shortest possible time. And the success story of communism in this respect during the past decade is bound to have a magnetic attraction that we dare not overlook. Moreover, as we well know, it is hard for these peoples to resist the communist-fostered view that all the institutions of the West are part and parcel of the colonialism from which they have just emancipated themselves.

These recently freed peoples and many others as well are determined to achieve as much industrialization as they can, and as quickly as they can. Otherwise they are doomed to remain indefinitely in an exposed position because as exporters of raw materials and importers of manufactured products they will be at the mercy of poor and fluctuating prices for their exports and inflation-prodded rising prices for their imports.

Also, we are beginning to fear that world-wide industrialization will leave us in a questionable competitive position for export markets because our mechanized production is based on a high wage level, whereas our developing competitors will have both mechanized production and a low wage level. We worry also because the rich domestic American market may become increasingly a prize to be sought by this formidable competitive combination. And the current American balance-of-payments situation gives ample evidence that this worry is well founded.

Hence, just past mid-century, we enter a new decade in a spirit of diminished national self-assurance. We are uncomfortable over the fact that in the years just past much of our policy has been a defensive reaction to the outward thrusting of Soviet expansionism. As we look over the scene, we know how hard it is—and for so many reasons—to seize the initiative in a way that will capture the imagination and cooperation of peoples who seem to be less concerned with ideology than with prosperity and material progress. We know that many of these peoples ascribe our great success less to the inherent excellence of our institutions than to our vast good fortune in having at our disposal land and mineral resources waiting to be exploited by a relatively small population. And so we wonder whether for the United States the next decade will be known in the future as the "Successful Sixties", the "Secure Sixties", or the "Slumping Sixties".

II

At this point one may wonder why such a discussion should bear the title, "Science, Politics and Peace". A moment's reflection should make the relevance clear. More than any other single factor, science and technology have influenced, even caused, these developments that have given us so much national concern. It was science and technology even more than ideology that enabled the Soviet rulers to weld the vast inchoate mass of the peoples of the USSR into a reasonably coherent body politic. It was science that enabled them to convert the vast territorial expanse of the Soviet Union from a strategic liability into a powerful asset. It was science that enabled them to give to their people enough material progress to keep them, if not happy, at least contented. And it was science, epitomized by developments in rocketry, that gave the added element of undeniable national prestige.

The same conclusion applies to the "underdeveloped" peoples. It was science, expressed through the media of modern communications, that gave hundreds of millions of desperately poor people a better knowledge of how other, more favored peoples, lived. It was science that gave them the hope of improvement through industrialization, better agriculture, and more education. Science gave them their higher level of expectation. And, of course, it also gave them the frightening problem of a burgeoning population as it reduced the death rate without bringing about any reduction in the traditionally high birth rate.

It was science that exposed the United States to the danger of attack from over the oceans of the world. It was science that brought about the shrinkage of the capability of the middle-sized states of the world, hitherto the Great Powers, into a permanently second-class status. It was science that enabled the unifying effects of a powerful ideology to bring China almost within a decade from a semi-colonial position to a world power of developing magnitude. It was, and is, science that has so profoundly and drastically altered the whole pattern and stuff of world politics and international relations.

Quite properly one may ask if science and technology have had any responsibility for the conditions that give us concern over the social and spiritual health of our own people. Here the answer is, of course, in the affirmative, though the relationship is less dramatic and obvious. Mass communications raise the lowest level of our culture but they also tend

to lessen the influence of the elite that set the standards of the past. Men in public life have less freedom from the immediate impact of poorly informed mass opinion than they had in the pre-scientific era. The economics of mass communication give to the citizen less exposure to divergent views, more exposure to a single view nationally carried to the most remote farmstead and hamlet.

Crime and juvenile delinquency cannot be disassociated from the rapid population growth in our urban centers, and this, in turn, results from the technologically produced increase in industrial employment opportunities and the declining need for a large rural population necessary for the production of our required foodstuffs. The family is becoming less effective as an instrument of education and social control because both parents and children are most scattered in their interests and activities. The home is reduced to a place where people eat and sleep and seek recreation in watching the dreary round of violence and brutality that nightly flickers across television screens. Our youth become the unconscious victims of a situation in which our moral resources are untaught or denigrated, while our weaknesses are magnified—even glorified. And we seem to lack the powerful and effective leadership that will mobilize our strength and minimize or hold in check our weakness.

III

As we search for guide-posts and solutions for these domestic and international problems, our first questions must be: What are the prospects that we will have a peaceful world in which to work them out? Prophecy is a risky business and few ordinary mortals have been notably successful at it. Nevertheless, some effort to estimate developing trends and various probabilities is as much a part of national life as it is in the life of an individual. Our great problem is to try to determine whether the destructive power now possessed by two or three states is likely to be used to plunge the world into chaos or whether it has become so terrifyingly lethal that the likelihood of its use becomes correspondingly remote. The essential facts are simple. Nuclear weapons are too risky for anyone to use unless their initial employment would be so crushing in its effect that the enemy state would be unable to strike a retaliatory counter-blow. No rational statesman could resort to an H-bomb attack unless he were sure on this point. But no such certainty exists at the present time, nor is it likely to exist in the forseeable future. Hence the

nuclear stalemate in which science, by making war so destructive, has actually created a climate favorable for peace.

There are, of course, remaining hazards. One is the possibility that a state with H-bombs might come under the rule of an irrational and megalomaniac dictator, of the type of Adolf Hitler, for whom rational considerations might be secondary. Another is the danger that local clashes involving minor states associated with nuclear powers might develop progressively until the major power on each side had become so committed to the protection of its status in the community of its allies that it might take a fatal step simply because the alternative seemed impossible and dishonorable to accept. The head of one of the great states of Europe recently told me that he regarded this hazard of gradual involvement as the greatest danger to world peace. I do not share this pessimism simply because, with the possible exception of Russian-Chinese relations, I do not believe the power of ultimate decision, or even control over the course of preliminary events, is likely to pass so completely out of the hands of the major power concerned. If this is true, we are left with the constant danger of local or "brush-fire" wars, but without the menace of great-power conflict, provided, of course, that the greatest powers maintain reasonable equilibrium in their atomic capabilities. Through its destructive power, science may have brought the world closer to peace.

But the cold war remains and it will not vanish, like a morning fog under the sunshine, after any summit conference or any series of conferences. The force of communist expansionism will remain vigorous for a long time, not merely because of ideology, but because it will continue to attract the millions of dissatisfied and restless people who make up the major portion of the earth's population. Even if all Soviet activities abroad were to cease, communism would still be a potent force for us to reckon with. And in the nature of things it must be expected that the Soviet Union will continue to look favorably on those who turn their eyes toward Moscow, as we do to our foreign friends and admirers.

Competition between Russia and the West will not be significantly moderated by victories by either side in the non-European world. It will be lessened in intensity and danger only when Russia and the great states of the West lose their mutual fear that each is out ultimately to destroy the other. This can only come about through gradual accommodation and understanding, and this process will be prolonged and

difficult. But there are some favorable straws in the wind. On our side, we now know that the Soviet system is not likely to collapse through inefficiency or bureaucracy or what we regard as unsound economics. On the other side, the Russians must know by this time that capitalism will not, as they once thought, collapse because of what they regard as its inherent contradictions.

It is easy to say that this mutual understanding cannot grow until the great specific problems, such as the division of Germany, Russian domination over Eastern Europe, and United States military bases in Europe, are resolved. It is my judgment that these specific problems will not be solved except as a consequence of a gradual lessening of mutual fear and hostility. This is why summit conferences are desirable. This is why even a limited degree of cultural and non-political exchange is desirable. Attitudes change more as a consequence of an aggregate of little events than as a result of great dramatic acts.

The United States will not maintain its position in the world, safeguarding its legitimate interests and assuring the maximum of security to our people, merely by possessing vast nuclear destructive power. This is commonly spoken of as deterrent capability, and it is not enough. Such power must be held by a people who are united in purpose, who are willing to work very hard for something more than money, and whose ideals are neither those of the cynical individual nor those of the marketplace. Somewhere along the line we have become fat, smug, and spiritually anemic. We interpret the "pursuit of happiness" to mean the pursuit of leisure, and for this reason we are at a loss when we try to explain our ideals to other peoples whom we would have copy our way of life.

Since our competition with other ways of life is a long-range affair, this spiritual flabbiness could be more dangerous to our survival than our failure to launch the latest rocket from Cape Canaveral. Other peoples before us have been wrecked by indolence, self-seeking, and a decay of national ideals. Most of us have read something of their history, and we have visited the ruins of their cities.

If we are likely to have peace—in the sense of relative freedom from the menace of atomic destruction—in the years ahead, but if we are certain to have a strenuous, competitive national life in which the fortunes of our country may rise to new heights or decay because of inner weakness, what can we do to protect ourselves for the future?

First, our people must somehow achieve greater discipline. Since ours is a democratic society, this must in large measure be self-discipline. But democracy and discipline are not inherently opposed to each other. Many other societies, fully as democratic as our own, lead a healthy, disciplined life that is not found with us.

Second, we need a massive reform in government. Discipline can never be achieved if public officials are corrupt and if laws are not enforced. Perhaps this is most important at the local level where the agencies of government most closely touch the individual, and particularly our young people. Even little things count. It must be hard, for example, to teach young people a proper respect for law when they see automobiles parked on the streets for days on end, and under NO PARKING signs.

Third, we need to make our system of public education far more rigorous and demanding. In many other democratic societies, schools and colleges are powerful social forces because they require far more than we do from their pupils and because they regard their function as education in the true sense of the term. Our public schools and far too many of our colleges have virtually abdicated their functions in society because they are content to give their students little more than an opportunity to have pleasurable social experiences.

If I emphasize the need for educational reform, this is not because I am a professional educator. It is because the world of the present—and still more the world of the future—is far too complex and difficult to be faced successfully by men and women whose precious youthful years have been spent in aimless pleasure or meaningless activity. Our competitors realize this need fully, and they are preparing to meet it. Unless we do as well—and I would hope, better—the years ahead, even if they be years of peace, will not be years of achievement and they will not be years of greatness. No people in history have been more favored by nature and circumstance. Let us hope that future historians will not decide that we were too greatly favored, and that we failed to justify the bounty lavished upon us. With direction, effort, and devotion to proper ideals we can make this next decade one of great achievement. If we do, the world will be the better off because of it, and so will we.

CITATION AND PRESENTATION OF THE ACADEMY MEDAL* TO EUGENE L. OPIE, M.D.

JOHN G. KIDD

Professor of Pathology, Cornell University Medical College; Pathologist-in-Chief, The New York Hospital

A MAN'S good fortune often begins before his birth, and so it seems to have been with Eugene Lindsay Opie. For he was born in Staunton, Virginia, almost 87 years ago—in a place, that is to say, where an established culture and the achievements of native sons gave reasons for pride, and in a time of peace and plenty. From his mother, Sallie Harman Opie, and his father, Thomas Opie—physician, surgeon, and teacher, one of the founders of The College of Physicians and Surgeons of Baltimore, and for 30 years Dean of its Faculty—Eugene Opie must have acquired those qualities of mind and character—searching curiosity, high intelligence, modesty, gentle humor, moderation, and uncommon good sense—that have distinguished him throughout one of the longest and most fruitful careers in American medicine.

Great as were his native endowments, they provided neither purpose nor means for Eugene Opie's work; but these were presently forthcoming, through determinations of his own. After receiving his bachelor's degree from The Johns Hopkins University in 1893, and attending Baltimore's College of Physicians and Surgeons for one year, he sought admission to advanced standing in The Johns Hopkins Medical School. His application was rejected at first on administrative grounds but later accepted, and in due course, with the others of the first graduating class, he received the M.D. degree in 1897. Largely influenced by one of his teachers—Dr. William H. Welch, then Professor

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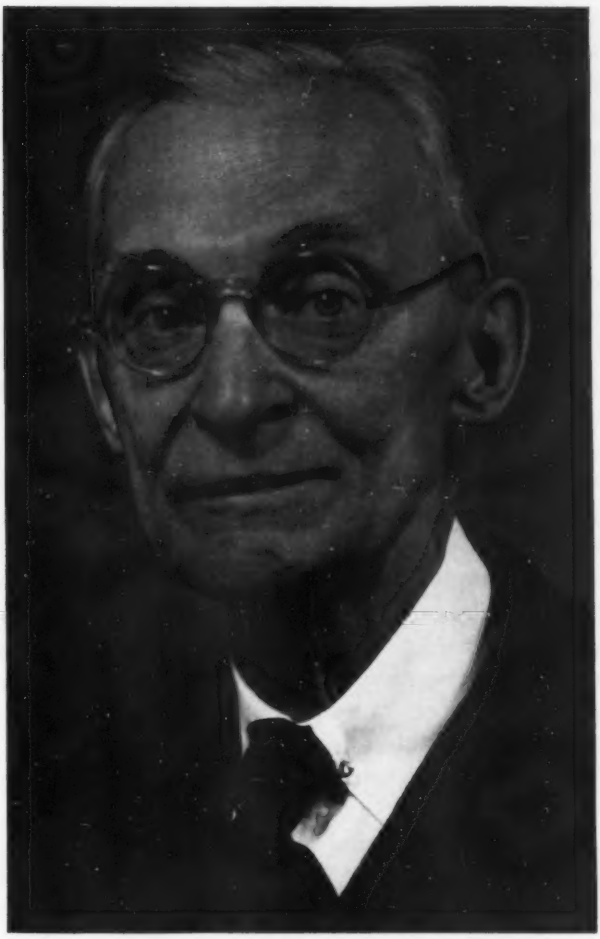


EUGENE L. OPIE, M.D.

of Pathology – Eugene Opie acquired, while still a medical student, what has become his dominant and enduring professional interest–the study of disease in the broadest sense of that term, which is to say, Pathology.

With this interest, and in his environment, it was inevitable that Opie should promptly have become a good microscopist. For through a succession of fascinating developments during the preceding 75 years, microscopy had then become what it remains to this day–an extraordinarily useful technique for those who would learn about the relations of structure and function in tissues and cells in health and disease; furthermore, two of his teachers, Osler and Welch, with remarkable perceptiveness, had introduced the microscope into the teaching of medicine in America, in 1877 and 1878, respectively. Acting upon a suggestion from Dr. William Sidney Thayer, who was then much interested in malaria, Eugene Opie and his classmate, W. G. MacCallum, made a search for malaria-like parasites in the birds of Baltimore during the summer following their third year in medical school. Their findings, exquisitely described and illustrated, were published in three papers in *The Journal of Experimental Medicine* in 1898. Opie described in detail the incidence and anatomical characters of protozoan parasites that he had seen in the blood of sparrows and red-wing blackbirds. He noted particularly the processes whereby certain of the microorganisms disrupted the corpuscles containing them and, escaping therefrom, became flagellated, the flagellae later disengaging themselves from the pigmented parent body and moving away in serpentine fashion through the plasma. His paper attests not only to his interest in and mastery of microscopy but also to his conservatism in interpretation.

Tools and techniques, however, were even then but strands in the string of Opie's bow. His teachers at The Johns Hopkins Medical School–notably Mall, Abel, and Howell in addition to Welch, Osler, and Halsted–had seen to it by various means, not least of which was precept, that the bow itself was properly fashioned, and of other materials. For these remarkable men, welded together as a Faculty, had transplanted from Europe, in a highly viable state and in an incredibly short span of years, something new to Medicine in the New World–namely, *a spirit of inquiry*, together with the essential means for furthering its ends: ceaseless questioning, careful observation, controlled experimentation, and critical reflection. Expertly laminated to suit his

purposes, these means and this spirit became Opie's bow. His arrows were his own, products of solitary work with senses, mind, and hands. Of the telling effects produced by some of these arrows, I shall now speak briefly—far too briefly to do them justice, it must be said, since the exigencies of taste, time, and occasion constrain us now as always.

Whenever we think nowadays of diabetes mellitus, or of carbohydrate metabolism, we immediately think also of those specialized pancreatic cells that make up the islands of Langerhans, and of the internal secretion that they produce. We are prone to forget, however —so vast and de-personalized has our store of medical knowledge become—that we make these important associations in large part because of discoveries made more than 50 years ago by Eugene Opie. While studying pathology in medical school, he saw the islands of cells, described some years before by Langerhans, in a section of pancreas handed to him by his teachers. Upon being told that nothing was really known about the nature and functions of the cells constituting the structures, Opie promptly undertook to learn what he could about them. His morphologic study, duly reported in *The Bulletin of The Johns Hopkins Hospital*, convinced Opie that these cells were highly specialized elements with an abundant blood supply, related embryologically to the parenchymal pancreatic cells but devoid of connections with the pancreatic ducts; from his own observations and from scrutiny of the previous work of Von Mering and Minkowski, and that of Ssobolew, he was prepared to believe that they might supply the internal secretion of the pancreas. In a correlated study, also begun while he was in medical school, Opie learned how to distinguish between two forms of chronic interstitial pancreatitis, in one of which the islands of Langerhans are conspicuously damaged. Some time thereafter, when he was a medical house officer of The Johns Hopkins Hospital, Dr. Opie was completing the postmortem examination of a person who had died from diabetes. With the aid of the microscope he saw that the islands of Langerhans had undergone degeneration and were now hyalinized! He grasped at once the implications of this new finding, and his careful study of additional cases soon provided convincing evidence that injury to the cells of the islands of Langerhans results in diabetes. These and Opie's further observations on diseases of the pancreas—notably his studies of the part played by pancreatic ferments in causing acute hemorrhagic necrosis of this organ under natural and experimental con-

ditions—made him an authority in this field within five years following his graduation from medical school. His book *Disease of the Pancreas: Its Cause and Nature*, now become a classic, was first published in 1902, the second edition appearing in 1910. For his observations on the causation of diabetes he was awarded the Banting Medal by the University of Toronto in 1946.

From his medical school days to the present, Opie has continuously inquired of Nature about disease. The results of his inquiries—freely given to medical science in two books, nine monographs, and more than 100 carefully written scientific papers—form a record of achievement which, impressive enough in bulk, is more impressive still when viewed in terms of depth, diversity, and importance. It was inevitable that his career—more than once termed peripatetic—should have been distinguished throughout. After six years in the Department of Pathology at Johns Hopkins, Dr. Opie was made a member of the newly founded Rockefeller Institute for Medical Research in New York—this in 1904, when he was 31 years old. During this, his first stay at the Institute, he served as co-Editor of *The Journal of Experimental Medicine*, for a time as Editor of *The Proceedings of the Society for Experimental Biology and Medicine*, and for three years as visiting Pathologist to the Presbyterian Hospital. In 1910, he became Professor of Pathology at Washington University, St. Louis, holding this post until 1923, and serving also as Dean of the Faculty during the period 1912-1915, and as a member of the Medical Reserve Corps, A.E.F., 1917-19. Appointed Professor of Pathology at The University of Pennsylvania and Director of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis in 1923, he remained in Philadelphia until he was made Professor of Pathology at Cornell University Medical College, and Pathologist to The New York Hospital, in 1932. Becoming Professor Emeritus in 1941, he has since carried on his work as Guest Investigator of the Rockefeller Institute. He was given honorary degrees by Yale University in 1930, Washington University in 1940, and his alma mater in 1947.

During his first period at the Rockefeller Institute, Dr. Opie worked mainly on certain fundamental aspects of inflammation, notably on the identification and effects of proteolytic enzymes of polymorphonuclear and mononuclear leukocytes in inflammatory lesions and exudates. Later he became interested in substances and conditions that bring

about necrosis of liver cells. Soon after going to St. Louis, he found that proper nutrition can preclude the harmful effects on the liver of alcohol, chloroform, and phosphorus given under experimental conditions. During and just after the First World War, in collaboration with Francis G. Blake, Allen Freeman, Thomas M. Rivers, and James Small, he studied two large epidemics of pneumonia following influenza in military camps, publishing the findings in book form in 1921. He began his studies on tuberculosis in 1908 during his first stay in New York, and carried them on until 1941 with increasing intensity at his successive posts in St. Louis, Philadelphia, and again in New York, as also during frequent excursions to the site of a Rockefeller Foundation study of tuberculosis in Jamaica, B.W.I., during the decade beginning in 1930, and on an extended visit to Peking, China, in 1939. In range, these studies have covered most of what we now know about tuberculosis, except for the effects of chemicals upon the disease. For example, the enzymes of tuberculous tissue and tuberculous exudates, the biology of the tubercle bacillus, cellular reactions of tuberculosis, and the fundamental phenomena of immunity and hypersensitivity in relation to the disease, have all engaged his attention, and to all these aspects of the larger problem his observations have brought new knowledge. But his broader and more important contributions were on the pathogenesis of the naturally occurring disease, with particular emphasis on the anatomical differences between first-infection and reinfection tuberculosis, on the means whereby tuberculosis spreads from infected to susceptible hosts, especially through family contacts, and on the implications for this disease of naturally occurring and induced immunity. It goes without saying that such knowledge has value, however judged. With good reason, he was given the Gerhard and Trudeau Medals in 1929, and the Weber-Parkes Medal and Award of the Royal College of Physicians in 1945.

Since his "retirement" in 1941, Dr. Opie has worked almost daily in his laboratory at The Rockefeller Institute. Studying first the nature of hepatic cancers brought about by feeding butter-yellow to experimental animals, he went on to a further evaluation of the dietary factors that influence the development of these growths. Then his attention turned to those formed bodies in the cytoplasm of hepatic cells which stain with basic dyes because they contain ribose nucleic acid. Noting that these bodies swell and lose their basophilism when the cells are

placed in water—or in "physiological" saline for that matter—he has carried on during the past several years an extensive study of the factors that determine the movement of fluids into and out of tissues and cells. Temperamentally incapable of being either a dilettante or an amateur, but by contrast always a responsible investigator in the real sense of that term, Dr. Opie has in the 18 years since his "retirement" systematically written up all his observations in the form of 16 scientific papers, each one complete and on a different theme; furthermore, he has submitted each of these 16 papers to a board of editors, who—judging its content critically against standards of soundness, originality, and significance, and having the obligation of rejecting the offering if it should prove faulty—have accepted it for publication. During this time he has also given his third Harvey Lecture — the only man so distinguished thus far.

One thing further must be said in relation to Dr. Opie's thought and work. Important as these have been and are, they have not by any means absorbed him. He has had a full, rich and joyful family life. And many a friend and colleague, young and old, having long enjoyed his generous companionship and depended upon his counsel, can testify that Eugene Opie's life has not been devoid of zest and fun. Great and good indeed has his fortune become!

Dr. Opie: For the depth and breadth and span of your continuing contributions to medical science, the President and Fellows of The New York Academy of Medicine now award you the Academy Medal. Long a beneficiary of your warmth and wisdom, I find great pleasure, and honor as well, in the privilege of presenting this Medal to you.

CITATION AND PRESENTATION OF THE ACADEMY PLAQUE* TO SHEPARD KRECH, M.D.

HOWARD REID CRAIG
Director, The New York Academy of Medicine

ONE of the pleasantest tasks which has been assigned to me over the years by the Academy's Committee on Awards has been the presentation of this plaque. Initiated eight years ago as the result of a bequest by the late Dr. Samuel McCullough, it is given as a token of recognition and appreciation of outstanding services and contributions to the life of the Academy. It should be of interest to recall the names of previous recipients of this award:

Orrin Sage Wightman, M.D.	Haven Emerson, M. D.
John W. Davis	Malcolm Goodridge, M.D.
Seth M. Milliken, M.D.	Charles C. Morchand
Montgomery B. Angell	

The plaque, of itself, is a small thing in comparison with the time, thought and energies which these men devoted to furthering the aims and effectiveness of the Academy in the fields of medical education, both lay and professional, public health, and the development of our library. Tonight's recipient has exhibited to an extraordinary degree these same traits of devotion and competence.

He was graduated from Yale College in 1913 and immediately entered the field of banking in which he continued until the outbreak of World War I, entering the Armed Forces as a member of Squadron A. He served in France as a detached officer and returned to this

* Presented at the Annual Meeting of The New York Academy of Medicine, January 7, 1960.

SHEPARD KRECH, M.D.

country after the Armistice in 1918, fully determined to go into medicine. He then entered Columbia University to pursue courses requisite for admission to medical school. The following fall he entered the Harvard University Medical School and graduated in 1923, just ten years after his graduation from Yale. Following an internship at the Presbyterian Hospital he devoted most of his time to the Columbia Division of Surgery at Bellevue Hospital. During the Bellevue period, with the encouragement of Dr. Ransom Hooker, he established a large and successful clinic devoted to patients with gastric and duodenal ulcer. It was also during this period that he published his widely recognized article on appendicitis. This article alone established his place in surgery. However, in 1938, at the age of 48 years, his career in surgery was cut short by illness from which, happily, he recovered but which precluded further activity in his chosen field. Since that time he has devoted himself to good works, the Academy being one of the chief beneficiaries of his interest and his energies.

For 21 years he served as an active and valuable member of the Committee on Public Health and in 1955 became an Emeritus Member of that Committee. In 1940 he became a Trustee of the Academy and in 1945 became Treasurer of the Academy, a position he holds at present. As Chairman of the Executive or Finance Committee of the Trustees, he has devoted himself assiduously to the responsibilities of that position. He has brought to the deliberations of that Committee an understanding of the problems and needs of the Academy, sound financial judgment, and an ability to get things done. The Academy is fortunate indeed to have a man of his caliber as its financial officer.

It gives me great personal pleasure and satisfaction to present to my good friend, on behalf of the Academy Committee on Awards, this plaque on which is inscribed:

PRESENTED TO
SHEPARD KRECH
IN RECOGNITION OF
OUTSTANDING SERVICE TO
THE NEW YORK
ACADEMY OF MEDICINE

FRACTURES OF THE SKULL: OPHTHALMOLOGICAL SIGNIFICANCE*

The Charles H. May Memorial Lecture

FRANK B. WALSH, JOHN W. MCMEEL AND ADOLF NEETENS

AN invitation to read a paper before The New York Academy of Medicine is an honor, and to be invited to give the May Lecture is a privilege. I am highly honored and deeply appreciative. I regret that I did not know Dr. May personally. I have enjoyed pleasant relations with the publishers of his book, the Williams and Wilkins Company of Baltimore. Through the courtesy of Mr. E. F. Williams I obtained a short statement which may interest you.

"Back in the year 1900 a young practicing ophthalmologist, Dr. Charles H. May, completed the manuscript for a manual of the diseases of the eye for students and general practitioners. He submitted it to a number of medical publishers but could not find anyone who would take it because he insisted that the book be sold for a price in the neighborhood of two dollars. In view of the fact that the book contained a number of color plates as well as many black and white illustrations, no medical publisher felt that it was a commercial possibility.

"Not to be daunted, Dr. May decided to take a gamble and invest his own funds in the manufacture of the book. This he did, and then persuaded the William Wood Company**, of New York City, to sell the book for him. The subsequent success of the book is well known to almost all present day medical men, for most of them have used May's *Diseases of the Eye* in their student days. And the actual property, the book itself, still belongs to the May family. They pay for the manufacturing cost of the book and furnish it to the publisher for sale on a royalty basis.

"In 1957 the 22nd American edition of the book was issued. It has

* Presented by Dr. Walsh, March 16, 1959, before the Section on Ophthalmology at The New York Academy of Medicine.
From the Wilmer Institute, The Johns Hopkins University and Hospital, Baltimore, Maryland.

** Purchased by the Williams and Wilkins Company in 1932.

been translated into some nine languages and at present a tenth is being added, namely Korean.

"Since the death of Dr. May the book has been carried on by his nephew, Dr. Charles A. Perera . . ." All of us have admiration for Dr. Charles Perera's resoluteness in keeping the book small and concise.

When one is invited to give a named lecture, the problem immediately arises as to a subject within one's capacity that might have value. In this regard I shall tell you something as to what influenced us to talk about trauma to the head. At the Wilmer Institute we have recently commenced a new project, primarily designed to add to our knowledge of the blood supply to the visual pathways in health and disease. Such a study has been made possible because we have obtained the active support of Dr. Richard Lindenberg, whose attainments as a neuropathologist are well known to all our colleagues in that field. A principal interest of Dr. Lindenberg's has concerned trauma to the central nervous system. His day by day activities in the Medical Examiner's office would readily be appreciated by those of you who have read *The Scalpel of Scotland Yard*[1], in which the more dramatic contributions of Sir Bernard Spilsbury have been recorded. Any Monday night session with Dr. Lindenberg exemplifies the type of observation that established Spilsbury as an authority in forensic medicine. While we have received help from Dr. Lindenberg, this introductory communication is not his responsibility. Several topics mentioned herein will be amplified in subsequent papers.

The ocular signs of trauma to the skull have been described in scattered communications insofar as the ophthalmologic literature is concerned. Because of the unusual opportunities we now enjoy, we propose to undertake a systematic study of trauma and its effects on the visual system. This paper represents an introduction to the subject and is the first of a series which is planned. In Part I of this communication, mention is made of pertinent anatomy and physics as concerns fractures of the skull. Part II is concerned with fractures in various regions, and our selection of material has been based on ophthalmological interest. The subject of skull trauma and its ocular signs is extremely complicated and in consequence various approaches to the subject might be utilized. Here we describe only injuries which may be associated with eye signs on the basis of fracture alone. Consequently, discussions

on coup and contrecoup, positive and negative pressure, shearing stress and herniations, as well as vascular changes, are left for subsequent communications. Furthermore, we are not here concerned with perforating injuries.

Part I

Anatomy. The effects of trauma on the skull are dependent in large part on the areas of strength and weakness of the skull itself and its supporting membrane, the dura, with its duplications, the falx and tentorium. The nature of the trauma is equally important.

The bony skull is composed of vault and base. The bones of the vault originate in membrane and those of the base in cartilage. The vault is more resistant to injury than the base, mainly because it has more elasticity. The vault has two bony tables, the outer and inner, which are separated by diploe. The vault does not contain foramina, which weaken the base. In consequence, fractures which involve only the vault are seen less often than those which involve only the base, and fractures which involve the vault tend to extend into the base.

There are areas of strength and weakness in both the vault and base of the skull. Rowbotham has described such areas of strength as arches of the vault and buttresses of the base. According to Dr. Lindenberg[2] the relative strength of the vault is principally explained on its configuration and elasticity, although there are arches that are stronger than adjacent areas. Such paired arches are situated on each side from the glabella to the external angular process, and from the mastoid to the external occipital protuberance. In addition, there is a strong ridge of bone running along each side of the longitudinal sinus from the glabella to the external occipital protuberance. The thin areas in the temporal region are strengthened by the overlying temporalis muscle. We have come across a diagram showing the areas of strength in the base of the skull according to the disposition of the medullary trabeculae such as occur elsewhere in the bony skeleton, where stress is maximal and strength is essential. The relationship of the indicated areas of strength, as shown in Figure 1—A, B, C, to the strong arches of bone in the vault does not require further comment, for in some areas, both anterior and posterior, the areas of strength are continuous.

The tentorium and the falx cerebri, which are merely reduplications of the dura, are supporting structures. They are important largely be-

Figure 1A—(left) Anteroposterior view of skull.

Figure 1B—(above) Skull—lateral view.

Figures 1A and 1B, and Figure 2, from Werner Spalteholz, *Hand-Atlas of Human Anatomy*, reproduced by permission of the publisher, J. B. Lippincott Company.

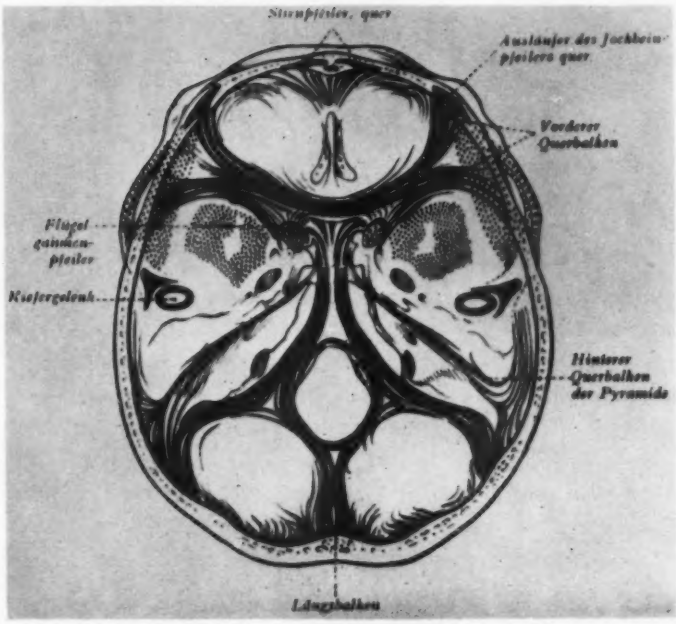


Figure 1C—Trabecular pattern base of skull (courtesy Dr. Lindenberg).

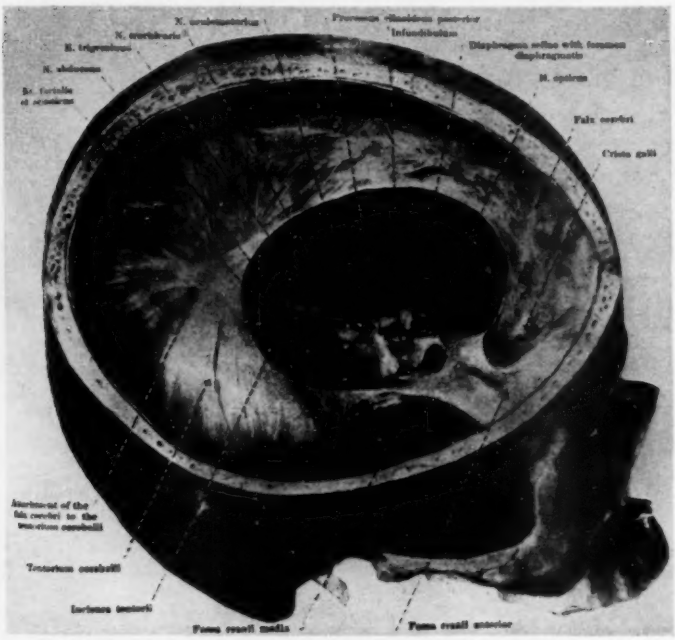


Figure 2—Drawing showing tentorium and falx (from Spalteholz).

cause of their relative immobility, which accounts for brain injury resulting from shearing stresses and pressure effects at their margins (Figure 2).

The bony orbits. The orbital cavity is bordered by thickened bone. The supraorbital margin is part of a strong arch of the skull, and is more prominent than the remainder of the orbital margin. The eyeball is protected by the bony margins of the orbit, and particularly by the bones of the nose, hence the frequency with which the nasal bones are fractured without the eye being damaged.

The roof of the orbit is formed by the orbital plate of the frontal bone anteriorly. This bone is thin and liable to fracture. The lesser wing of the sphenoid forms the apex posteriorly, the optic canal being formed by the union of its two roots. The trochlear fossa is situated in the anteromedial angle of the orbit, and injuries in this area account for superior oblique involvement.

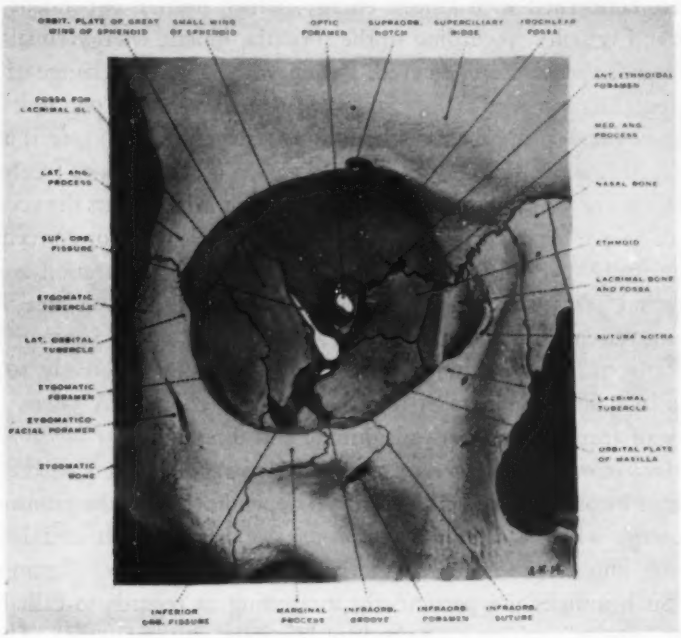


Figure 3—The orbit from in front.

Reproduced from Eugene Wolff, *Anatomy of the Eye and Orbit*, 2nd edition, 1940, with the permission of the publisher, H. K. Lewis & Co. Ltd., London.

The lateral wall of the bony orbit is composed of the orbital surface of the malar bone over its anterior one-third and the greater wing of the sphenoid in its posterior two-thirds. This wall of the orbit is separated from the superior and inferior walls by the superior and inferior orbital fissures respectively.

The floor of the orbit is composed of the orbital plate of the maxilla, the zygomatic bone and the orbital process of the palatine bone.

The medial wall of the orbit is composed of the lamina papyracea of the ethmoid, the angular process of the frontal bone, the lacrimal bone and the lateral aspect of the body of the sphenoid. The medial wall of the orbit is even thinner than the roof of the orbit (Figure 3).

Physics. An understanding of possible results of trauma to the skull demands reasonable knowledge of certain basic physical phe-

nomena. *Trauma* is dependent on force. *Force* is a form of energy; here we are concerned with kinetic energy, which itself is dependent upon mass and velocity, according to the formula: kinetic energy equals one-half mass times velocity squared. *Velocity* is the rate of change of position per unit time. *Acceleration* is change in velocity per unit time. *Deceleration* is properly described as negative acceleration. *G* is a constant which is a basic measure of acceleration that applies to freely falling objects in the earth's gravitational field, and it represents the acceleration of 32.2 feet per second per second; the term is used for convenience in the study of head trauma as a measurement of acceleration and deceleration. *Stress* is a measurement of force applied per unit area; it can be a measurement of push or pull or of a shearing stress. *Shearing stress* results from two contiguous parts of a body sliding relatively to each other in a direction parallel to their plane of contact. A *vector* is an entity of force having both magnitude and direction.

Mechanisms of fractures of the skull and orbit. Impacts on the skull result in local or general deformations depending upon the intensity of the energy which is absorbed and on the size of the impact area. Injuries of lesser impact will not produce fracture but may produce brain damage. Such injuries are particularly interesting as regards so-called "indirect injuries", which may produce blindness in the absence of either unconsciousness or fracture. Injuries to the skull may be divided into two large categories, a) non-perforating injuries and b) perforating injuries. The size of the impact area is important as regards perforation; obviously, the smaller the impact area, the greater is the likelihood of perforation from any given force. Non-perforating injuries may be due to 1) low-velocity impacts like, for example, automobile accidents, and 2) high velocity impacts like, for example, non-penetrating bullet wounds or hammer wounds.

All injuries are the result of absorption of energy, and three conditions may influence injury to the head: 1) The head may be at rest but free to move when it is struck by a moving object; in this instance the head will be accelerated. 2) The object may be at rest and be struck by the moving head; in this instance there is deceleration of the head. 3) A moving object may strike a supported and resting head; here there is a tendency toward excessive damage to the skull rather than to the brain.

Absorption of energy in injuries to the head produces 1) contusions

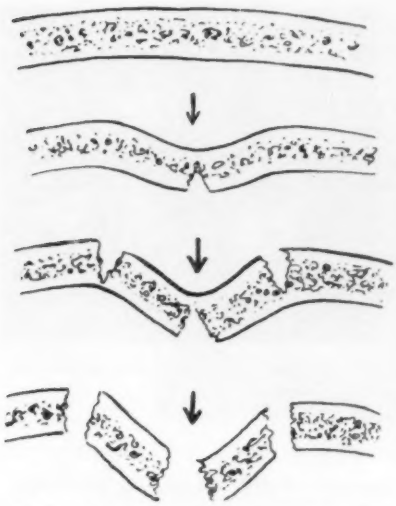


Figure 4A[3]—Diagram illustrating mechanism of skull fracture.

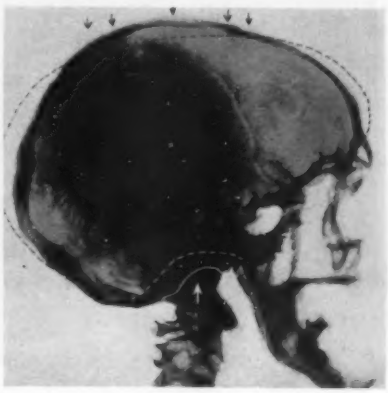


Figure 4B[3]—Diagram illustrating skull distortion.

Figures 4A and 4B reproduced from G. F. Rowbotham, *Acute Injuries of the Head*, with the permission of the publisher, E. & S. Livingstone, Limited, Edinburgh.

or laceration of the face or scalp; 2) deformation of the face or skull; 3) increased pressure within the cranial cavity, the increased pressure being due to the deformation of the skull and acceleration or deceleration of the head. In the instance of wounds to the skull, much energy is absorbed by the scalp, particularly with glancing blows. Deceleration is made less abrupt when there is resilience or "give" to the object with which the head has impact. For example, an individual may be thrown headfirst through a windshield, which absorbs energy by shattering, with the result that there is no skull fracture or, indeed, in some instances no real injury. In the same manner dashboard padding, added as a safety device, may serve to supply resilience and minimize injury.

A blunt object moving at a low velocity, such as a brick or baseball striking the skull, or as in an automobile accident when the skull strikes the windshield or dashboard, is likely to produce two different types of fractures: 1) there may be a depressed fracture at the site of impact, or 2) a linear fracture which is located away from the site of impact or has its inception away from the site of impact. Linear fractures occur

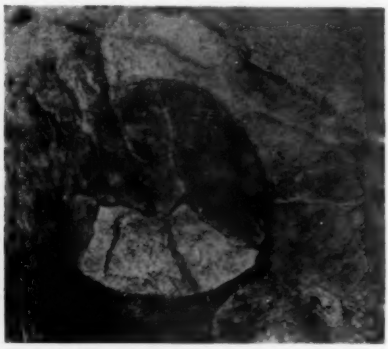


Figure 5A (left) and 5B (right)—Photograph of stellate fracture and curvilinear fracture. (A) from without; (B) from within.

at areas of stress produced at the junction of the inbending and outbending areas of the skull that are present when the impact occurs (Figures 4 A, B[3] and 5 A, B). When the force is of lesser intensity, only one linear fracture may occur. This naturally is in the area of greatest weakness (area of primary stress level). If the force of impact is greater, a second and third linear fracture may result. Although the radial type of linear fracture begins at the junction of inbending and outbending, the fracture line may extend to the site of impact because this is the area of stress concentration. At the site of impact there is first fracture of the inner table, whereas away from the site of impact the outer table is first to be fractured. When the object is less blunt and its velocity is greater, there is a tendency toward the production of a curvilinear fracture about the site of impact. Sometimes two or three concentric fractures may be present in such injuries. When the object is large and its velocity is low, and when the mobility of the skull is absent or slight, as for example a wagon wheel passing over a head, there is likely to be distortion of the skull without involvement of the brain rather than fracture of the skull (Figures 4 A, B, 5 A, B and 6).

Several generalizations as regards fractures are made at this time. Messerer stated that longitudinally or transversely directed forces tend to produce fractures in the corresponding axes. Aran showed that many fractures of the vault are vertical and continuous in the adjacent basal fossa. These observations have been established (Rowbotham[3]). Basal

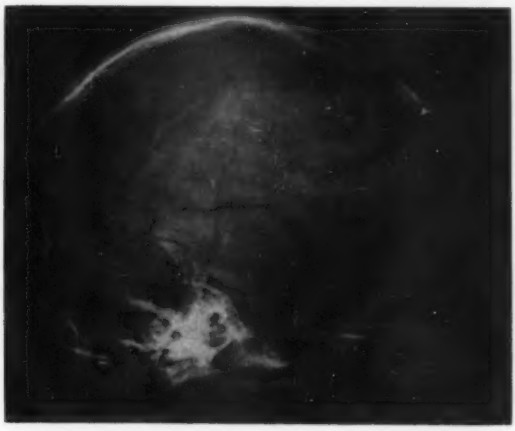


Figure 6—X-ray stellate fracture, parietal region.

fractures also may occur as a result of direct injury to the base, by general distortion of the skull and by force applied to the base through the spinal column (for example, falling on the buttocks), or force applied to the face. Lissner and Evans[4, 5] have stated that blows in the midline occipital region generally produce basal fractures. A posterior parietal blow in general produces fracture in the temporal region. A midfrontal blow produces fracture above or through the orbits. Blows to the frontal region, to the vertex and to the temporal region are likely to result in fracture extending into the floor of the anterior cranial fossa, and less often into the middle fossa as well. Trauma to the posterior skull tends to produce fractures extending into the posterior and middle fossae but not to the anterior fossa. According to Gurdjian and Webster[6] one half of linear skull fractures involve the middle third of the skull and tend to extend to the middle fossa; the remaining half are about equally distributed between the frontal and occipital regions. Depressed fractures in half the cases are in the frontal region (Figures 7, 8, 9 and 10).

Fractures of the orbit. Since the floor of the anterior fossa is made up of the roof of the orbit, we shall consider here fractures of the orbit and anterior cranial fossa, as follows: 1) direct injury to the frontal region; 2) direct injury to the facial region; 3) direct injury to the

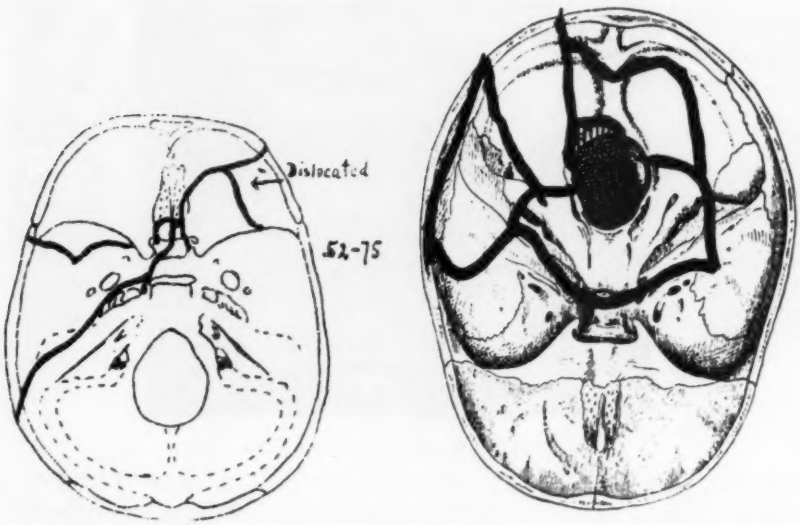


Figure 7—(left) Fracture resulting from motor cycle accident. Site of impact was right frontal region. Fracture through roof of orbit extending into the middle cranial fossa, of opposite side. Longitudinal fracture of petrous bone.

Figure 8—(right) Diagram illustrating fractures involving posterior and middle fossae. Trauma to occipital region as result of fall.

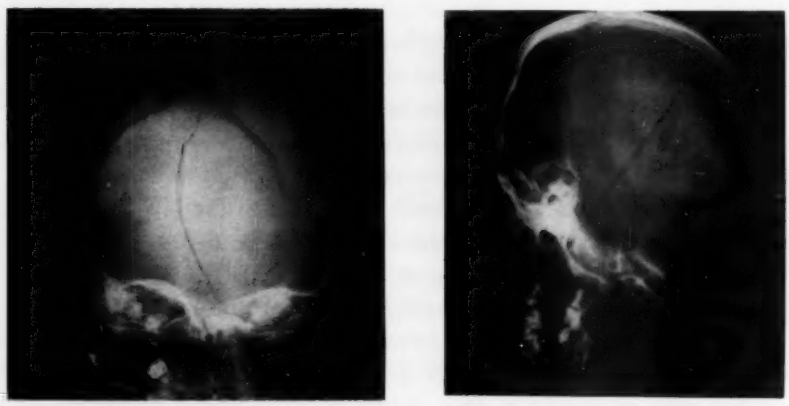


Figure 9—(left) A. Fracture, occipital bone extending into posterior fossa. Patient an infant.

(right) B. Fracture, parietal bone extending into middle cranial fossa. Patient an infant.

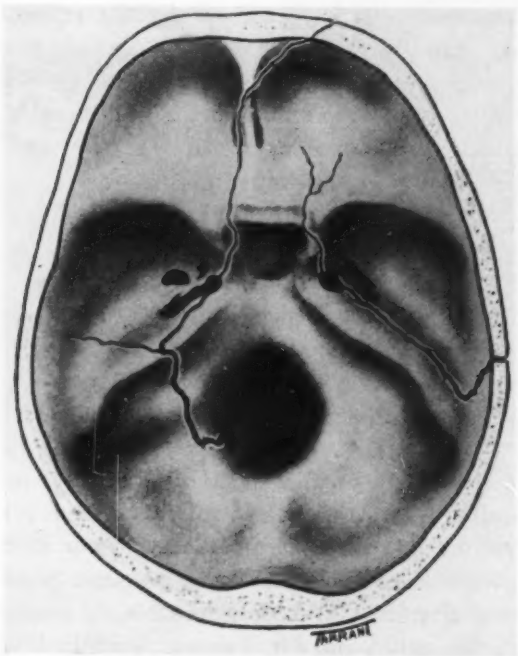


Figure 10—Diagram of typical fracture lines of the base of the skull showing involvement of the orbital roof.

Figures 10, 11 and 12 reproduced from Duke-Elder, *Textbook of Ophthalmology*, vol. 6, with the permission of the publisher, Henry Kimpton, London.

orbit; and 4) indirect orbital injuries, that is, injuries that have extended into the orbit from elsewhere, or result from injury to the skull at a remote site, usually the occipital region. This last-named type is a contrecoup fracture and occurs extremely rarely.

1) *Direct injury to the frontal region.* This is rather often characterized by fracture at the external or internal frontal processes. With or without fracture of the superior orbital rim, there is likely to be fracture of the posterior portion of the orbital roof because of its relative thinness. Such fractures may be associated with cranial nerve palsies and loss of vision. The cranial nerve palsies are readily explained by involvement of the superior orbital fissure. Defects in vision are explained by involvement of the optic nerve because it is available for

injury as it passes through the optic canal. If the force is greater the fracture may extend posteriorly and involve the region of the sella turcica (Figures 7 and 10). In some instances extensive fractures of the bones of the orbit do not account for recognizable involvement of the nerves and do not affect vision. Furthermore, there are cases in which vision is depressed when no fracture is made out. Such cases are further described subsequently. In some instances, injury to the frontal region produces fracture of the base of the orbit as an isolated involvement or in association with fracture of the roof.

Particular mention is now made of fracture into the optic canal. Ever since Lillie and Adson[7] reported their two cases, we have been looking for a duplicate case in which callus formation within the canal would produce a change in the visual fields as proof that there had been a fracture into the canal. No such case has been encountered by us. Recently I wrote Dr. C. Wilbur Rucker regarding his experience with fracture of the optic canal at the Mayo Clinic. He replied that he and Dr. J. Grafton Love have not seen any such case. It is Dr. Rucker's belief that fracture into the optic canal must occur extremely infrequently. Dr. Raymond Pfeiffer has stated the same belief. Dr. James Watts observed fracture of the optic canal with a spicule of bone entering the optic nerve, and Dr. Lawton Smith observed this in a patient he saw in Boston. In neither instance was there recovery of vision.

2) *Direct injury to the facial region.* Le Fort described three lines of weakness in the bones of the face as are shown in Figure 11. a) The commonest fracture is present in most extensive lesions involving the face. Starting from the lower part of the nasal cavity, the line crosses the canine fossa and runs up and backwards to the pterygomaxillary fissure. The fragment involved compromises the alveolar border, the vault of the palate, and the pterygoid process. This fracture is without particular ophthalmologic interest. b) This line of weakness runs across the midline of the face, leaving the zygomatic arch uninjured. It involves or runs below the inferior part of the nasal bone, crosses the frontal process of the maxilla and the lacrimal bone, usually above the nasolacrimal canal, traverses a variable and sometimes extensive area of the floor of the orbit, which shatters without offering much resistance, crosses the lower orbital margin in the region of the maxillozygomatic suture (leaving the zygomatic bone unbroken), traverses the infra-

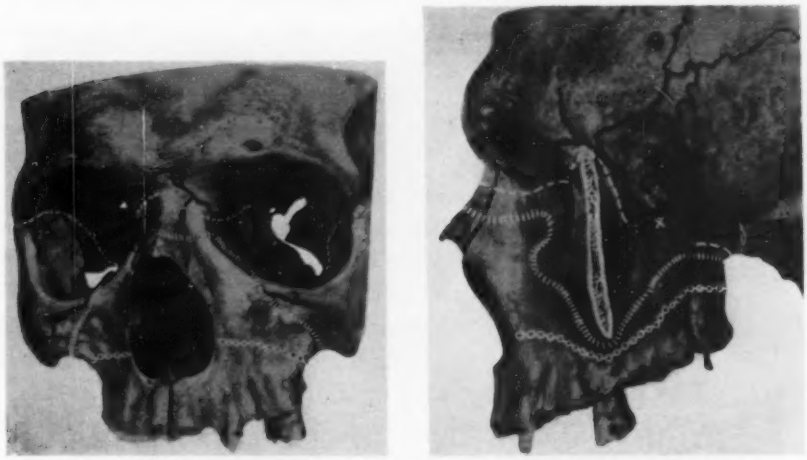
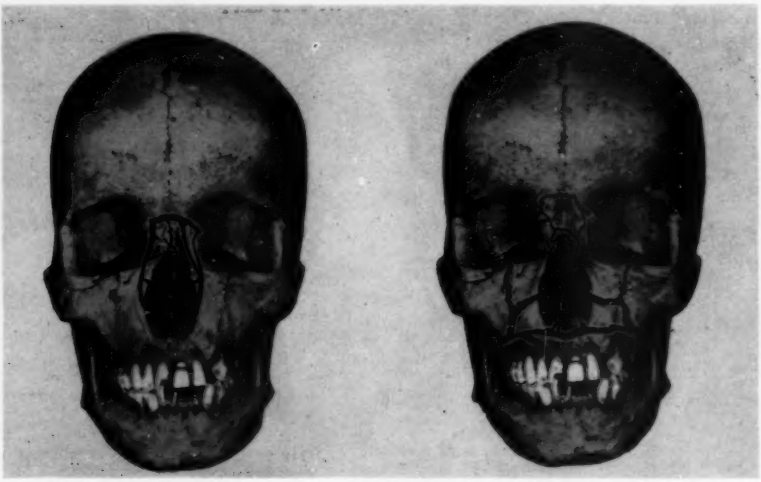


Figure 11—The fracture lines of LeFort, A (left) anterior; B (right) lateral.

orbital foramen, and fractures the pyramidal process of the maxilla and the pterygoid process, to end in the pterygomaxillary fissure. In a complete fracture the fragment thus involves part of the nasal bones, the lower part of the frontal process of the maxilla and part of the lacrimal bone. c) In its essentials this line of weakness allows a separation of the face from the cranium above the zygomatic arch. The line starts through the upper part of the nasal bone; the upper cranial extremities of these bones are very resistant indeed, and if these give way, the fracture line traverses the cribriform plate and the floor of the anterior fossa. From the nasal bones the line runs through the medial wall of the orbit in the neighborhood of, or below, the frontomaxillary suture, the upper part of the lacrimal bone, across the ethmoid, which is extensively fissured so that the ethmoid cells are exposed, and then descends below the optic canal (which escapes) to the infraorbital fissure, where the line of weakness bifurcates, one arm running upward and forwards, separating the roof of the orbit from its lateral wall, to terminate just below the frontomalar suture at the external angular process, the other running downwards and backwards from the pterygomaxillary fissure to divide the pterygoid processes near their bases. The bulk of this fragment therefore consists of the mass of the zygomatic arch (Duke-Elder)[8].



Central naso-maxillary fracture, first degree.

Central naso-maxillary fracture, second degree.

Central naso-maxillary fracture, third degree.

Lateral malar-maxillary fracture (after A. McIndoe).

Figure 12

Nasomaxillary and malar fractures. Direct injuries to the face such as occur in automobile accidents are likely to produce nasomaxillary fractures of varying extent. Duke-Elder's figures (Figure 12) exemplify what may occur directly to the face with relatively mild and more severe traumas. Such injuries account for deformities of the nose and face.

3) *Direct injuries to the orbit.* The rim of the orbit is relatively strong, and consequently in many direct injuries to the orbit the rim remains intact. Fracture of the roof has been mentioned. Fracture of the medial wall is commonly associated with emphysema. Fracture of the floor of the orbit is particularly important in that it is likely to be associated with subsequent enophthalmos.

4) *Indirect orbital fractures.* Probably such fractures are most often seen as the result of fracture of the base extending to the bones of the orbit. According to Duke-Elder, they extend either along the lamina cribrosa between the inner and middle third of the orbital roof, or temporally, involving the lesser wing of the sphenoid. Such fractures tend to run alongside the optic canal and rarely extend into it. The roof of the orbit may be fractured by contrecoup, with the site of impact being at the base or at the occipital region, in which instance bilateral fractures of the orbital roof may occur. Blindness following fracture of the orbit produced by a fall in a sitting position has been described. Generally speaking, injury to the orbit is likely to produce fracture of the inner wall and base of the orbit, whereas injury to the frontal area is likely to produce fracture of the roof of the orbit.

Part II

Topical diagnostic features of fractures of the skull and orbits. The signs of fracture of the orbit now commented upon are: exophthalmos, enophthalmos, emphysema, gross hemorrhage, "black eye", cranial nerve palsies, loss of vision and intraocular changes.

Exophthalmos. After an injury to the face, the eye may be pushed forward either as a result of hemorrhage into the orbit or from air reaching the orbit. It may result from inflammation incident to the injury. We have seen exophthalmos result from the introduction of water into the orbit when the maxillary antrum was irrigated after the floor of the orbit had been inadvertently punctured. Fracture of the floor of the orbit may be associated with bleeding, which produces exophthalmos,

and after the blood has been absorbed, enophthalmos may be the end result. It is not always possible to demonstrate fracture extending into the orbit when orbital hemorrhage occurs. In some such instances the fracture, although present, cannot be identified. In other instances the orbital hemorrhage has occurred, in all probability, from some other mechanism such as strangulation of the ophthalmic vein, possibly as a result of pressure from the cavernous sinus. Dr. Lindenberg observed at autopsy fracture of the bones of one orbit in which there was absence of hemorrhage, but the other orbit, where no fracture could be demonstrated, contained blood. We have observed massive hemorrhage into the orbit in newborns, and this has ultimately disappeared spontaneously without residual effects. Orbital hemorrhages in both orbits were present in an infant who also suffered from intracranial bleeding; this child succumbed. We have observed bilateral exophthalmos associated with injury to the face and hemorrhage into the orbit: suturing the margins of the eyelids was necessary to protect the corneae. Particular mention is made of hemorrhage into the roof of the orbit when such hemorrhage has been subperiosteal. It may appear as an orbital tumor, and later make its true nature known by hemorrhage appearing subconjunctivally 24-48 hours after the accident.

Enophthalmos. Enophthalmos following trauma is now generally believed to be due to fracture of the floor of the orbit. In some instances the fracture is not seen on x-ray. There is prolapse of orbital fat. Usually the enophthalmos is not noted until several weeks after the accident has occurred. We have notes on a patient with enophthalmos who was shown to have had prolapse of orbital fat into the ethmoid sinuses. Mention has already been made of exophthalmos following trauma being due to hemorrhage. If the orbital floor has been fractured, enophthalmos may be the ultimate result. Pfeiffer[9] pointed to the true etiology of traumatic enophthalmos. In 120 fractures of the orbit, he found only the floor affected in 24 cases. He suggested that impact of the eyeball on the floor of the orbit might in some instances account for the fracture.

Emphysema. Air from the nasal sinuses may gain access to the orbit as a result of fracture of the lamina papyracea of the ethmoid or from fracture of the floor of the orbit. Crepitation is almost invariably noted. The eye may be shoved back into the orbit without any real sense of resistance, even though it may be proptosed. Exophthalmos associated with emphysema is characterized by narrowing of the lid fissure, where-

as exophthalmos from other causes usually is associated with widening of the fissure.

The "black eye". Differentiation between a black eye, the result of a direct blow, and what occurs after fracture of the skull is important in diagnosis. Hamilton Bailey's differential diagnosis between orbital hemorrhage consequent upon a fractured anterior cranial fossa and a "black eye" cannot be improved upon. 1) In fracture of the anterior cranial fossa, the extravasated blood is limited sharply by the palpebral fascia to the orbital margin. It tends therefore to be circular. In "black eye" there is no such limitation. 2) In fracture of the anterior fossa the discoloration is purplish at the commencement, while the "black eye" is beefy red from the start. 3) In "black eye", when there is a conjunctival hemorrhage, it is in the conjunctiva and is moved when the conjunctiva is moved. When there is a fracture, the hemorrhage is subconjunctival. 4) In a conjunctival hemorrhage associated with a black eye, there is a posterior limit to the extravasation, while in the fractured anterior fossa there is no such limit; also, in the latter, the hemorrhage tends to be fan-shaped, with the handle of the fan toward the iris. The butterfly distribution of the blood staining is characteristic in our experience; Blakeslee[10] discovered eyelid and conjunctival hemorrhages in 17 per cent of his 610 cases of fractured skull. Disposition of the hemorrhages varied. Conjunctival hemorrhages were temporally situated rather often and could be considered as hematomas in some instances. He found many cases in which fracture was far from the orbit. In his series of cases, 20 per cent of those who exhibited orbital hemorrhages died. In our experience hemorrhage into the lids or conjunctiva is usually associated with fracture either of the orbit or the anterior fossa, rarely from distant fracture.

Cranial nerve palsies. Cranial nerve palsies, so frequently associated with trauma to the head, may be associated with orbital injury and are here considered in that regard. If the ophthalmoplegia is total it can be stated with assurance that the nerves have been affected at the level of the superior orbital fissure or cavernous sinus. If, in addition to the first and second divisions, the third division of the fifth nerve is affected, there would of necessity be intracranial involvement because the third division does not reach the orbit. Orbital damage may account for palsy of muscles supplied by the superior division of the third nerve (superior rectus, levator) or of the muscles supplied by the inferior division of

the nerve. The site of involvement of the fourth or sixth nerves may be orbital but often cannot be definitely identified as such. Pupillary dilatation may be associated with other evidence of third nerve involvement. It may occur with an isolated involvement of the inferior oblique muscle or from a direct blow to the eye. Orbital injury may account for apparent paresis of the superior oblique muscle when the trochlea has been dislocated. Trauma may account for ptosis, with or without associated superior rectus palsy.

Encephalocele. This is an occasional complication of fracture of the roof of the orbit. King saw a patient in whom, as a result of injury, an encephalocele had invaded the orbit and ethmoid sinuses so that brain tissue could be seen protruding into the posterior nares.

Mucocele. This is sometimes an end result of injury to a sinus. Such mucoceles usually originate from the frontal sinus but may so originate from the ethmoid sinuses.

Pneumocephalus is a collection of air in the cranial cavity. It results from a cranionasal or cranioaural fistula.

Cerebrospinal fluid rhinorrhea is another complication of cranionasal fistula in a majority of cases; also it may result from a fistula extending into the middle ear, producing an otorrhea.

Injury to the eyeball. Here mention is made only of optic nerve avulsion, which is usually caused by perforating injuries such as a pick being driven into the orbit and pulling the optic nerve out of the eyeball. We have seen optic nerve avulsion when the eye was struck by a fist and the optic nerve was forced out of it in the manner that a cork is removed from a bottle by striking its butt on the wall.

Injury to the optic nerve. In many cases injury to the optic nerve results from direct damage to the nerve, such as results from knife or ice pick wounds. However, in a majority of cases of injury to the nerve, there is absence of fracture, and loss of function is the evidence that the nerve has been injured. In such cases there may or may not be fracture of the orbit or skull and there may or may not be unconsciousness at the time the injury occurred. We have notes on a single case of direct injury to the optic nerve by a spicule of bone forcing its way into the nerve (courtesy of Dr. James Watts).

Here we are concerned with indirect injuries to the optic nerve. The description of Turner[11] has seemed to us to be accurate and has applied to our cases.

The injury may be trivial or severe. Often the forehead, the supraorbital ridge or the external angular process is the site of trauma. Injury to the posterior aspect of the skull rarely accounts for damage to the optic nerves or chiasm. The loss of vision is immediate. Rather often there is some improvement within a few days after the accident, and the vision sometimes returns to normal within three or four weeks. If recovery of vision has been made, it is extremely rare that it deteriorates subsequently. There is a tendency toward central scotomas and sector defects in those cases where the loss of vision has been incomplete. The pupil on the side of the injury is often somewhat dilated and sluggish to light. The optic fundi may remain quite normal or a few retinal hemorrhages may be present. In many instances, whether or not there is recovery of vision, there is the development of pallor of the optic disc commencing about three weeks after the injury. Papilledema is notably absent in such cases.

As regards the etiology of loss of vision in such cases, it would seem probable that the site of injury is at the level of the optic canal, thus explaining the development of pallor three weeks after injury. The problem immediately arises as to the nature of the injury. Turner has suggested, and we are in agreement, that in all probability there is interference with the blood supply to the nerves. It seems unlikely, but possible, that intraneural hemorrhages could be responsible.

Hemorrhage into the optic nerve sheaths. At one time it was frequently assumed that hemorrhage into the optic nerve sheaths was a cause of loss of vision. Such an assumption is not justifiable. When hemorrhage occurs in the optic nerve sheaths there may well have been concomitant damage to the nerve itself. We have observed individuals in whom there was shown to be massive hemorrhage within the optic nerve sheaths, and yet there had not been prior to death any pronounced loss of visual acuity. It is not established that the pressure of hemorrhage within the sheaths is responsible for change in visual acuity.

Fracture of the sellar region and rupture of the chiasm. From what has been stated regarding changes in the conformation of the skull, it is obvious that anteroposterior compression could account for tearing separation of the bones and the base of the skull and fracture at the region of the sella turcica. If the lateral diameter of the skull is suddenly widened, the optic chiasm can be put on stretch. We have observed such a case and have a section showing an actual division of the chiasm itself (Figure 13).

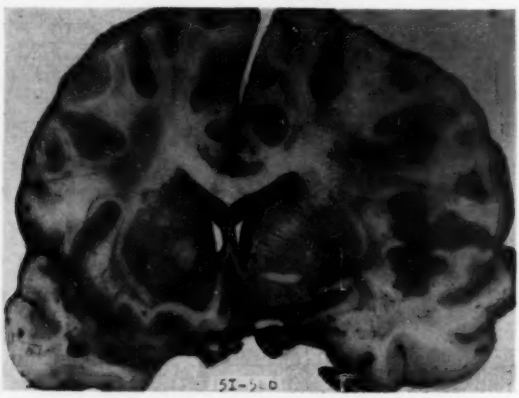


Figure 13—Rupture of chiasm. Extensive basal fracture. Chiasm divided by bone fragment.

It seems established that traumatic bitemporal hemianopsia is associated with demonstrable fracture in a minority of cases. Best studied 315 case reports of bitemporal hemianopsia and in only 13 instances was fracture of the skull demonstrable. In possibly 30 per cent of cases the vision of one eye has been lost and there is a temporal defect in the field of the opposite eye. Osterberg[12], also Henderson and Rucker have suggested that bitemporal hemianopsia is usually due to microscopic ruptures of the small nerve fibers in the crossing bundles. Traquair, Dott and Russell[13] felt that in the majority of cases, small pial vessels supplying the chiasm were ruptured, and that this accounted for the hemianopsia. They remarked that such patients often exhibited hypothalamic symptoms such as sleep disturbance, temperature regulation disorders and diabetes insipidus. As described by François, Neetens and Collette[14], occlusion of the central artery to the chiasm might explain bitemporal hemianopsia. Generally speaking, patients with bitemporal hemianopsia resulting from trauma do not regain the lost fields.

The cavernous sinus syndrome. This syndrome is seen occasionally as a result of trauma when a fracture of the base may have been demonstrated. In the fully developed syndrome there is total ophthalmoplegia with dilated fixed pupil, loss of corneal sensation and some proptosis as well as venous engorgement of the eye on the homolateral side. The cavernous sinus of one or of both sides may be affected as the result of

fractures which involve the sellar region, an area of relative weakness. Usually the trauma has been directed to the vertex or to the frontal region. We have seen such unilateral involvement immediately after injury. Dr. Lawton Smith observed total bilateral ophthalmoplegia and bilateral blindness in a patient who had sustained severe skull trauma. Whereas direct involvement of the cavernous sinuses by hemorrhage accounts for the ophthalmoplegia, the explanation for the visual loss is not entirely clear. In some such instances it is possible that there has been concomitant indirect injury to the optic nerve (or nerves). We have observed a single example of cavernous sinus thrombosis resulting from injury, and since aseptic cavernous sinus thrombosis is rare, the case is briefly summarized:

> T.D. (#15462), a 66 year old man, fell on the floor. He was unconscious when first seen and remained so for two days. He developed total ophthalmoplegia on the right. Six days later a subdural hematoma was evacuated from the right side. He lost hearing in the left ear after the fall and had blood escape from that ear. During his stay in the hospital he developed paresis of the left sixth and third nerves, but these palsies cleared and the movements of the eye became normal. Several months later there was pronounced recovery as regards the right ophthalmoplegia but he had developed the syndrome of aberrant regeneration of the third nerve on that side. (Courtesy Dr. Maurice Silver.)

Carotid cavernous fistula. Trauma is responsible for this lesion in approximately 75 per cent of cases. It is the only traumatic intracranial anteriovenous aneurysm. It is the only intracranial site where an artery passes through a venous sinus. Fracture is not necessary where trauma is the exciting cause. Proptosis of the opposite eye is a common development, and this is explained on the basis of engorgement of the opposite cavernous sinus through the intercavernous sinuses.

Generalizations regarding trauma and injuries to nerves 3-6th, inclusive. These and other intracranial nerves may be injured a) by fracture through the foramen or canal through which the nerve passes, b) bruising of the nerve against the bone, c) disruption of the blood supply to the nerve, d) local pressure caused by hemorrhage or edema, e) torsion of the brain causing avulsion of the root. It is often impossible to distinguish the mechanism which is responsible.

Damage to the third, fourth and sixth nerves in closed head injuries usually results from injuries involving the orbits, less commonly from injuries involving the cavernous sinuses. In a minority of instances, stretching of the nerves as the result of displacement of the brain stem accounts for the nerve palsy. It might be anticipated that trauma, par-

ticularly trauma to the base, would account for sixth nerve paralysis more often than third nerve paralysis because of the long course of the sixth nerve intracranially. However, this is not the case. Turner found that in his large series the third, fourth and sixth nerves were involved equally often. The third nerve is available for injury at the tentorial edge and also to pressure between the posterior cerebral and superior cerebellar arteries. The sixth nerve is not in a position to be injured by the tentorium, but is in a position to be injured directly at the petrous apex. It is thought that sixth nerve paralysis is often due to increased intracranial pressure when tumor is present, and this would seem to apply after trauma when the sixth nerve defect develops after a delay. Isolated fourth nerve palsy is a rare occurrence after injury; in all probability some such cases remain undiagnosed.

The fifth nerve is rarely injured intracranially. When it does occur, the gasserian ganglion has usually been injured as the result of a basal fracture crossing the tip of the petrous bone. Injuries to the frontal bone and the facial bones may result in branches of the first and second division being interrupted.

Following injury to these nerves, regeneration occurs in most instances, providing that the injury is to the peripheral nerve. When the nerve supplies several muscles, as in the instance of the third and seventh nerves, so-called aberrant regeneration or "misdirection of regenerated fibers" may occur and result in a characteristic syndrome.

Fracture of the temporal bone. These fractures are important in that they are associated with traumatic paralysis of the seventh and eighth nerves. Whereas such paralyses often occur together, we shall first consider peripheral seventh nerve paralysis.

The seventh nerve is commonly paralyzed by injury which has caused a fracture extending down from the temporal region into the petrous pyramid. The facial nerve is particularly vulnerable either at the internal auditory meatus or in the second portion of the fallopian canal. If the ear drum is ruptured there is escape of blood from the ear, and if the dura is torn there will be escape of spinal fluid as well. There may be, in addition to the seventh nerve paralysis, loss of the sense of taste, and loss of lacrimation and salivation on the ipsilateral side. Such a longitudinal fracture causing facial palsy often does not account for coincident loss of eighth nerve function. In longitudinal fractures of the petrous bone, facial paralysis occurs in 10-18 per cent (Grove[16]).

Peripheral facial palsies occasioned by injuries peripheral to the point of emergence of the seventh nerve from the stylomastoid foramen are not considered here. In cases of facial paralysis, regeneration of the nerve may occur producing so-called mass movement of the face (aberrant regeneration). The development of "crocodile tears" seems best explained on the basis of aberrant regeneration of this nerve (fibers designed for the salivary glands reach the lacrimal gland of that side, and vice versa). Bilateral facial paralysis following fractures of the temporal bone are uncommon. Facial paralysis immediately following trauma has a poorer prognosis than such paralysis when it occurs after an interval following the trauma. As a point of interest, we have never had to do a tarsorrhaphy on any patient who spontaneously developed Bell's palsy, but a tarsorrhaphy has been almost always necessary after a removal of an angle tumor.

Fracture of the petrous bone is likely to produce deafness and labyrinthine signs. Damage to the auditory nerve occurs much more often than to the vestibular nerve. Whereas a longitudinal fracture of the petrous bone may produce labyrinthine signs (dizziness, vertigo, nausea, diplopia, nystagmus), such signs almost always indicate transverse fracture of the bone. It is in this type of fracture that the labyrinthine bony capsule is fractured, whereas it is often spared in the longitudinal fracture. Transverse fractures of the petrous bone usually are a part of severe injury, and often the patient does not survive. If recovery occurs, the nystagmus, which is horizontal and rotary, clears after several weeks. Whereas longitudinal fractures usually occur as a result of trauma to the temporal region, transverse fractures usually result from trauma to the occipital region. Although the petrous bones are considered as strong areas of the base, transverse fractures involving them are not uncommon, when the stress is perpendicular to the bone.

Conclusions

Because this is an introductory paper, and in subsequent communications topics here mentioned will be amplified, no effort is made to summarize what has been stated. However, several generalizations are offered:

1) Trauma to the skull or orbit may result in ocular symptomatology whether or not an associated fracture is present.

2) The mechanics of skull fracture have been outlined briefly.

3) Trauma to the anterior skull and orbit are likely to produce ocular symptomatology, whereas trauma to the posterior skull rarely does so.

4) Fractures in themselves are relatively unimportant. It is the injury that is associated with fractures which is significant.

5) In this paper no mention has been made of coup and contrecoup, shearing stresses, herniations and vascular lesions, all of which remain to be considered in subsequent communications.

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THE HEMODYNAMIC CONCEPT OF ATHEROSCLEROSIS*†

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INTRODUCTION

THE hemodynamic concept of atherosclerosis considers the laws of fluid dynamics as the primary factor in the pathogenesis of atherosclerosis. The laws of fluid mechanics are applied to the natural conditions in the circulatory system. Hydrodynamic factors can account for the localization of atherosclerotic lesions at specific areas of predilection and its absence in apparently identical adjacent areas of intima. Local factors, based upon the laws of hydrodynamics, can account for the inception, the progressive changes and the varied pathologic appearances of arterial occlusive disease in the aorta and coronary arteries as well as in arteries of the viscera and extremities.

The contributing atherogenic effects of other factors, such as age, sex, race, diet, nutritional status, habitus, lipid metabolism, drugs, hormones, associated diseases, hypertension and stress require further evaluation. It is suggested that these factors may modify the primary hemodynamic mechanism.

FLUID DYNAMICS

The motion of fluids may be streamline or turbulent. In streamline or laminar motion the fluid moves in definite layers or paths. In turbulent motion the fluid moves in an eddying mass and motion at a given point varies irregularly from instant to instant. At low velocity the motion of fluid is usually streamline. As the velocity is increased the motion

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† From the Department of Forensic Medicine, New York University Post-Graduate Medical School and the Office of the Chief Medical Examiner, City of New York.

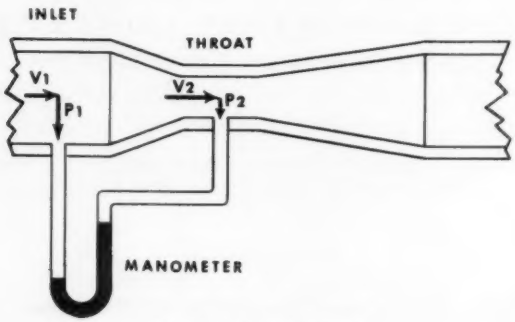


Fig. 1—Venturi meter.*

*Figures 1, 2, 4, 5, 6 and 8, from: Texon, M. A hemodynamic concept of atherosclerosis with particular reference to coronary occlusion, *A.M.A. Arch. intern. Med. 99*:418-27, 1957, reprinted by permission.

breaks down and becomes turbulent. When lines of flow converge, as in the arterial tree generally, or in an area of narrowing due to an atherosclerotic plaque, there is a tendency toward stability or streamline flow. Flow in such tubes with converging boundaries or narrowed lumina is characterized by an increase in velocity and a decrease in static pressure. These forces are the basis for Bernoulli's Theorem which states that fluid in motion possesses energy by virtue of its velocity and its pressure. The sum of the static pressure and the square of the velocity is constant if fluid flows from Point 1 to Point 2 and if there is no loss of energy between these two points. Thus, as in a Venturi meter (Figure 1), flow in a tube with converging boundaries causes the lateral pressure to be reduced at the narrow portion where the velocity is increased. In curvilinear motion (Figure 2), the lateral pressure is increased along the outer curvature by virtue of the centrifugal force. The lateral pressure on the inner curvature is decreased in direct proportion to the same centrifugal force. Finally, since the velocity of flow at a cross section of a tube (Figure 3) increases from the wall toward the center, division of the more central or axial fluid stream at a site of bifurcation or branching results in a relative increase in velocity and a relative decrease in static pressure at the medial walls of the crotch zone.

Physical factors formulated in Bernoulli's Theorem are the basis for such diversified practical applications as flight in the air due to the lift of an airfoil (Figure 4) and innumerable hydraulic devices utilizing differences in pressure or suction effects. Similarly, the physical factors based on Bernoulli's Theorem clarify the predilection for atherosclerotic lesions to occur at areas in the circulation characterized by converging boundaries or tapering, branching, bifurcation, abrupt curvature and external attachment.

Hydraulic Conditions

The circulation may be considered as a hydraulic system with unique specifications or hydraulic conditions. The hydraulic conditions are determined by a number of factors, as follows:

1. Physical characteristics of the blood
   - (a) Viscosity
   - (b) Specific gravity = s, or density = ρ.
   - (c) Homogeneity (particulate size)
   - (d) Temperature
2. Flow characteristics
   - (a) Velocity of flow = V = cm/sec.; Velocity pressure = $\rho\frac{V^2}{2}$
   - (b) Lateral pressure = p = dynes/cm.^{2}
   - (c) Volumetric rate of flow = Q = cm.^{3}/sec.
   - (d) Pulsation of flow
   - (e) Pulse rate, rhythm and amplitude
   - (f) Reynold's number = R = $\frac{VD}{\mu}$
3. Anatomical pattern (geometry of design)
   - (a) Caliber of lumen = D = cm.
   - (b) Tapering
   - (c) Bifurcation
   - (d) Branching characteristics
   - (e) Curvature
4. Fabric or local mural factors
   - (a) External attachment
   - (b) External pressure
   - (c) Thickness
   - (d) Elasticity
   - (e) Porosity
   - (f) Strength of bond between layers

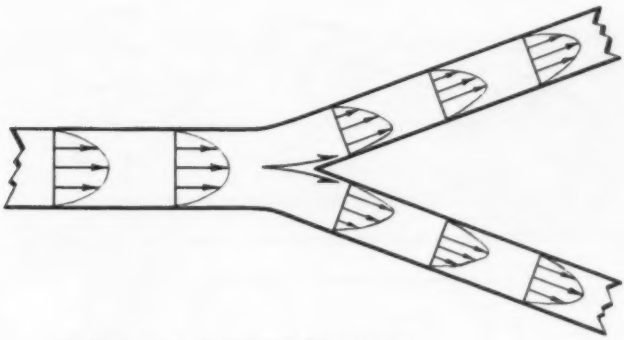
Sites of Predilection, Localization and Progressive Changes

The operation of the laws of fluid dynamics under conditions found in the circulatory system is prerequisite and conducive to atherosclerosis. The earliest lesions are found at certain sites of predilection. These are areas characterized by curvature, bifurcation, branching, external attachment or tapering. Such locations are subject to a relative decrease

Fig. 2—Elbow flow meter.

in local lateral pressure in accordance with the laws of fluid mechanics *(vide supra)*.

The decrease in lateral pressure at points of predilection produces, in effect, a suction action. The intima is here disposed to a lifting or pulling effect upon the endothelial layer and superficial cells. This represents the initial change. The local reaction consists of a reparative process, a thickening due to the proliferation of intimal cells and fibroblasts from the subjacent layers. The thickened intima gradually projects into the lumen, thus creating an increased Venturi effect. The lateral pressure becomes further decreased as the caliber of the lumen diminishes and progressive changes appear. Cellular elements and lipids are added to the intimal proliferation and fibrosis. The vessel presents an eccentric, or rarely concentric, thickening of its intima corresponding to the composite effects of the local hydraulic conditions of flow, curvature and attachment. The thickened intima assumes the well defined appearance of an atherosclerotic plaque. The pathology involves chiefly the intima from its endothelial layer to the basement layer adjacent to the media. These changes include proliferation of intimal cells, fibroblastic proliferation and deposition of collagen, elastic tissue changes, cellular infiltration, lipid infiltration and occasional calcifica-



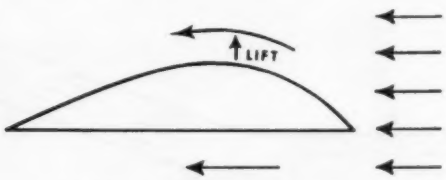
VELOCITY DISTRIBUTION FOR LAMINAR FLOW
ALONG A TUBE AND AT A BIFURCATION

Fig. 3—Cross section velocity of flow.

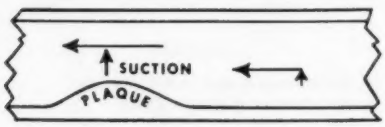
tion. Vascularization of the intima also occurs through the growth of capillaries from the lumen or from the adventitia.

The continued operation of the local hydraulic factors makes further pathologic changes possible. These may be stationary for long periods, slowly progressive or episodic in nature. A critical stage in the pathology may arrive when a relatively quick or dramatic change takes place: 1) Shearing off or lifting of the superficial layers of an atherosclerotic plaque may occur. A raw or ulcerated surface becomes exposed to the flowing blood elements. These may become deposited to form a thrombus. The thrombus may enlarge to a partially or totally occlusive degree by the accretion of additional blood elements. 2) An intramural or intimal hemorrhage may result from the same hydraulic forces which tend to draw the plaque toward the lumen. The intima is split or torn locally to produce a microscopic intimal hemorrhage in a manner comparable to a gross medial dissecting hematoma of the aorta. Intimal hemorrhages in association with atherosclerotic plaques are frequent. They vary in size; a small hemorrhage may not be sufficient to affect the caliber of the lumen while a large hemorrhage in association with an atherosclerotic plaque may reduce an already narrowed lumen even to the point of total occlusion.

The pathology inherent in atherosclerosis produces occlusive changes of all degrees as the net result of composite local hemodynamic factors. Partial to complete occlusion may result locally from 1) extreme



AIRFOIL LIFT DUE TO INCREASED AIR VELOCITY



VELOCITY AND PRESSURE CHANGES (SUCTION) ASSOCIATED WITH ATHEROSCLEROTIC PLAQUE.

Fig. 4—Similarity of dynamic factors involving an airfoil and an atherosclerotic plaque.

intimal thickening, 2) thrombosis superimposed upon the ulceration of an atherosclerotic plaque, 3) intimal hemorrhage or hematoma in association with an atherosclerotic plaque and 4) intimal hemorrhage associated with thrombosis. Partial to complete occlusion distally may result from embolization of the atheromatous content of a plaque by rupture of the overlying intima as it is drawn toward the axial stream.

Illustrative Examples

(a) The coronary circulation
(b) The aorta
(c) Bifurcation of the aorta — common iliac arteries
(d) Splenic artery
(e) Pulmonary artery
(f) Veins

(a) *The Coronary Circulation:* The coronary arteries are unique with respect to their hydraulic conditions. The flow of blood in the coronary arteries is intermittent as a result of systolic contraction. The stream is subject to rapid changes in velocity, increased flow occurring during diastole and reduced flow occurring during systole. The coronary arteries appear to be unique in the body with respect to such phasic variations in blood flow. It is noteworthy that the caliber of the

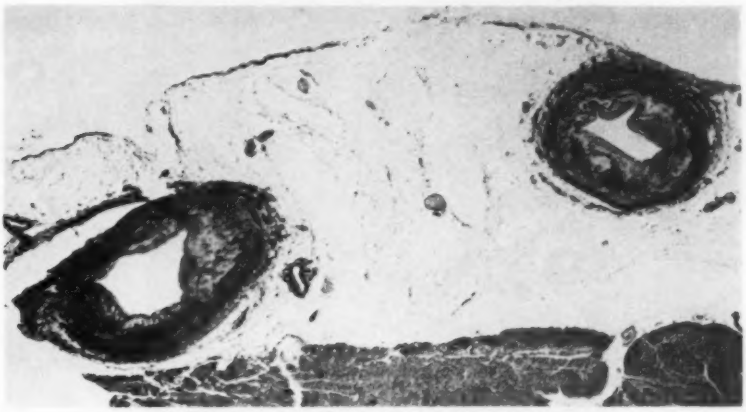


Fig. 5—Left coronary artery, anterior descending branches. The "converging boundary" lesion produces concentric atherosclerosis; "branch" and/or "curvature" lesion produces eccentric atherosclerosis.

extramural coronary arteries tapers rapidly. The anatomical curves inherent in the coronary circulation are also notable. The composite effects of these factors, namely, the rapid changes in velocity of flow, the nozzle effects of tapering and the inherent anatomical curvatures appear to be significant factors in the predisposition of the coronary arteries toward atherosclerotic changes. Pathological examination of coronary arteries reveals uniformly that the atherosclerotic process develops at sites of predilection which are determined by the local hydraulic conditions. A free and straight vessel shows correspondingly more concentric atherosclerosis (Figure 5). The nature of flow in a zone of sharp curvature determines a greater degree of involvement of the internal radius in comparison with the external radius (Figure 6). The free epicardial arc of an involved coronary artery may be less affected than the attached myocardial arc. The reduced lumen at the site of atherosclerotic change due to curvature or attachment will be, of necessity, eccentrically placed. A frequent finding is a linear atherosclerotic plaque involving the left coronary artery beginning approximately 1 cm. from its origin at a point where it curves and continues to form the left anterior descending branch. This is aptly described as a "waterfall lesion".

Fig. 6—Atherosclerosis of the internal wall of the right coronary artery in the A-V sulcus near the acute border of the heart—a "curvature" lesion.

(b) *The Aorta:* The predilection of the aortic arch for atherosclerotic changes and dissecting hematomata appears determined by the increased velocity and reduced static pressure or suction effect in this region. The sudden release of intra-abdominal pressure in the Valsalva maneuver causes a sudden increase in aortic blood velocity. The accompanying suction effect may tear the aortic wall and produce a dissecting hematoma (Figure 7). A common finding is a large atherosclerotic plaque on the inner curvature of the aortic arch, a "sentinel patch". One of the earliest plaques to appear is located at the obliterated exit of the ductus arteriosus, an "attachment" lesion. The aorta typically and invariably presents atherosclerosis predominantly on the dorsal surface. The tapering of the aorta results in a relative increase in blood velocity in the abdominal portion with consequently more severe atherosclerosis in this area (Figure 8). In advanced instances of atherosclerosis

Fig. 7—Dissecting hemorrhage (hematoma) of the aorta.

Originally published in Burchell, H.G. Aortic dissection (dissecting hematoma; dissecting aneurysms of the aorta), *Circ. 12*:1068, 1955. Reproduced by permission of the author.

of the aorta, the involvement may be diffuse, affecting both dorsal and ventral walls.

(c) *Bifurcation of the Aorta–Common Iliac Arteries:* The bifurcation of the aorta divides the more central or axial blood stream. Under such hydraulic conditions a relative increase in velocity and a relative decrease in static pressure are produced at the medial walls of the crotch zone. These forces, as described above, determine these areas as sites of predilection for atherosclerotic changes (Figure 9). It is noteworthy that the proximal margin of the atherosclerotic change in the common iliac arteries may be about 1 cm. distal to the bifurcation in the early stage of development. It appears that this area is free of atherosclerosis due to the local increase in pressure due to impingement of the blood stream. The impingement pressure is less prone to produce

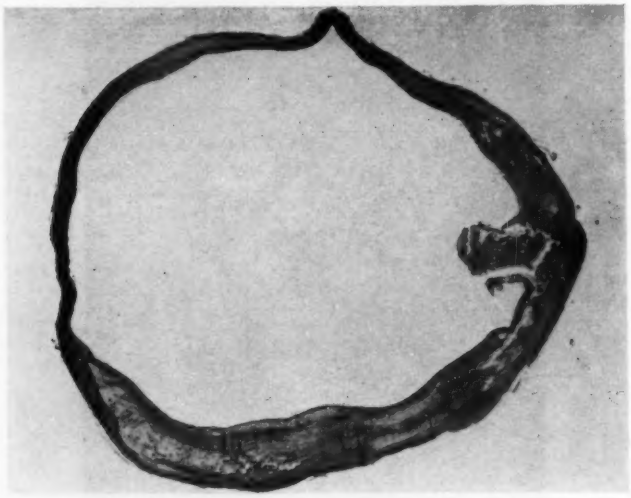


Fig. 8—Atherosclerosis of the dorsal wall of the abdominal aorta—an "attachment" lesion.

atherosclerosis than the diminished lateral pressure in the immediately distal crotch zone.

(d) *Splenic Artery:* The splenic artery is remarkable for its tortuosity and relatively large caliber. The large caliber and tortuosity serve to decrease the blood velocity. Atherosclerosis is therefore relatively infrequent. Atherosclerosis is, nevertheless, found as a result of the local hydraulic conditions in accordance with the laws of fluid dynamics. An atherosclerotic plaque is noted on the first inner curvature in the specimen illustrated (Figure 10).

(e) *Pulmonary Artery:* The pulmonary ring and adjacent pulmonary artery have a greater circumference than the aortic ring and ascending aorta. Since, normally, the same blood volume per unit of time must pass these orifices, it is obvious that the velocity of flow is diminished in the pulmonary artery. This is associated with a relative increase in lateral pressure. Therefore, atherosclerosis due to the suction effect of the stream is minimized in the pulmonary artery.

(f) *Veins:* In veins, which generally present diverging lines of flow and have a larger caliber, the velocity is relatively low and the static

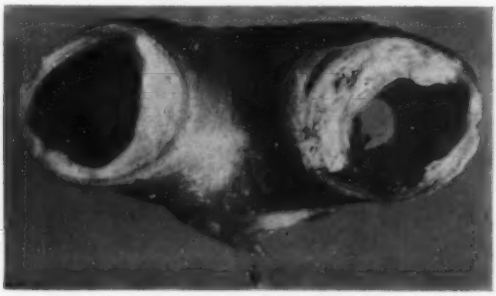


Fig. 9—Atherosclerosis of the medial walls of the common iliac arteries—a "bifurcation," "Y," or "crotch" lesion.

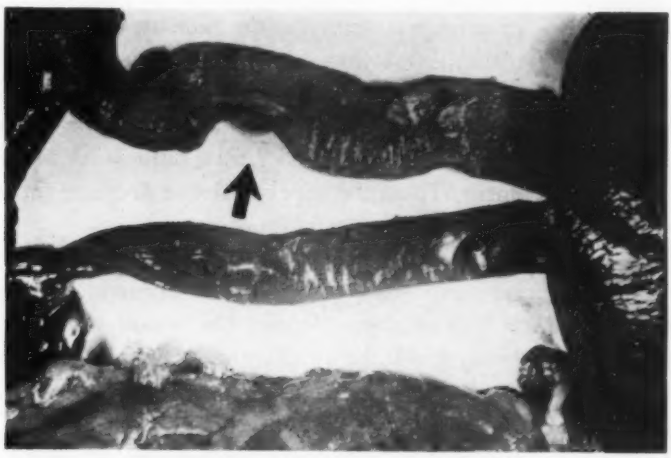


Fig. 10—The splenic artery showing tortuosity and "curvature" lesion.

pressure is relatively high. The suction action upon the walls of veins is therefore minimal, and occlusive sclerotic changes are comparatively rare.

Discussion

The hemodynamic concept of atherosclerosis considers the laws of fluid dynamics as the primary factor in the pathogenesis of atherosclerosis. The concept accounts for the localization and progressive changes

found in arterial occlusive disease in the aorta and coronary arteries as well as in the arteries of the viscera and extremities. The absence or a minimal degree of sclerosis in the pulmonary artery and in veins is also accounted for on the basis of hemodynamics.

The variations in the severity of atherosclerosis found in individuals appear to be due principally to individual differences in their hydraulic specifications. The velocity of flow, the caliber of the lumen and the anatomical pattern seem to be of utmost importance. A biological factor must also be considered. This is the capacity for local reparative reaction by the intima. It is here that genetic differences in tissue or fabric structure and differences in biological response to injury may determine the nature and degree of atherosclerotic change.

Summary

Hemodynamics has been presented as the primary factor in the development of atherosclerosis. A physical basis for occlusive arterial disease is offered. The laws of fluid mechanics determine the predilection for atherosclerotic lesions to occur at areas in the circulation characterized by converging boundaries, abrupt curvature, bifurcation, branching and external attachment. These are sites of relative decrease in lateral pressure in accordance with Bernouilli's Theorem.

The hemodynamic mechanism is described for intimal ulceration, intramural hemorrhage and dissecting hematoma occurring as complications in the pathogenesis of atherosclerosis. The hemodynamic concept applies to the circulation in the aorta and coronary arteries as well as to the arteries of the viscera and extremities. It also applies to the pulmonary artery and the venous circulation generally.

The hemodynamic concept considers the velocity of blood flow and the nature and biological response of the tissues to be closely associated with the variations in degree of atherosclerosis among different individuals. The principal cause of variation in the severity of atherosclerosis appears to be due primarily to differences in hydraulic specifications. Additional factors are described. These may merely modify the primary hemodynamic mechanism. Atherosclerosis appears to be a sequel primarily of fluid dynamics applied to the natural conditions in the circulatory system.

A Bibliography may be found in Texon, M., A hemodynamic concept of atherosclerosis with particular reference to coronary occlusion, *A.M.A. Arch. intern. Med.* 99:418-27, 1957.

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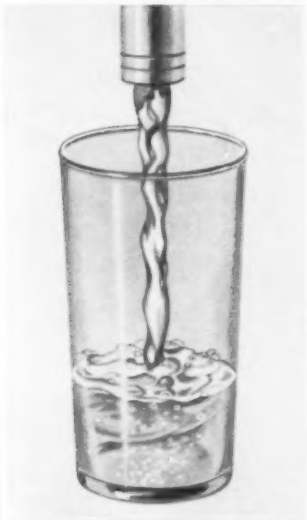

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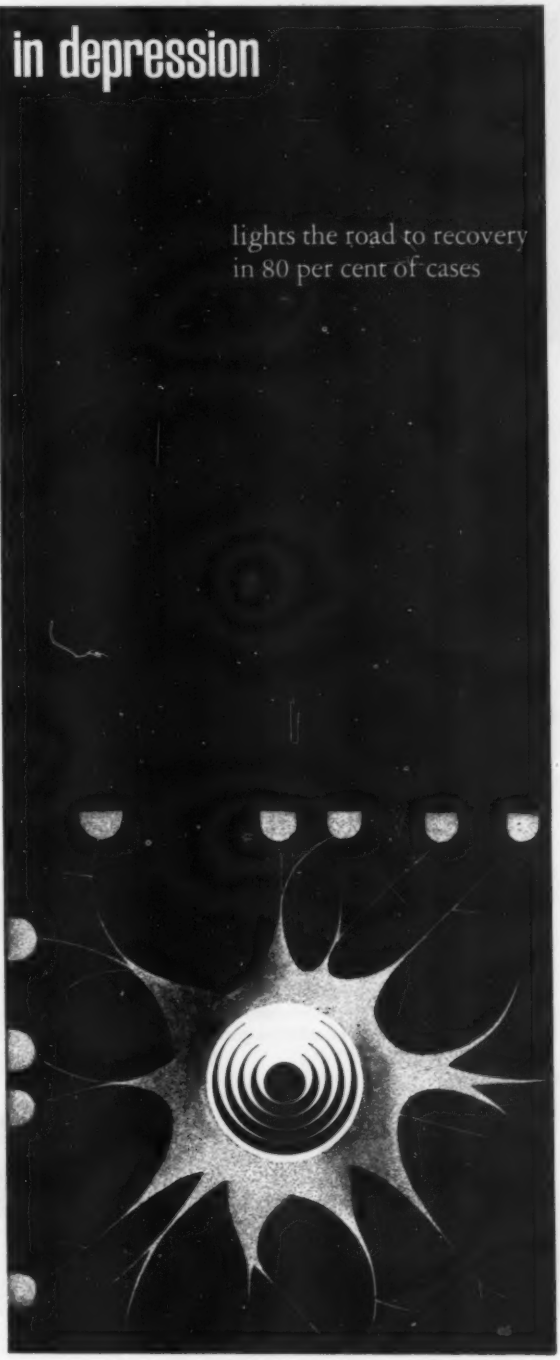

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